THE WRITINGS OF
R WILLIAM BROADBENT

WALTER BROADBENT

OXFORD MEDICAL PUBLICATIONS

WRITINGS OF SIR WILLIAM BROADBENT

Medical and Neurological

Edited by

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SELECTIONS FROM THE WRITINGS

Medical and Neurological

OF

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INTRODUCTION

By his son Walter Broadbent

In publishing this volume my object has been to make a selection from the numerous papers written by Sir William Broadbent, without including any of those used as the basis of his books on *The Pulse* and *Heart Disease*. I have inserted a few written since on cardio-vascular subjects, among them his last completed paper on "The Examination of the Heart." The papers have been chosen for various reasons, some for their historic interest, such as the one on the Bilateral Association of Nerve Nuclei, called by Dr. Hughlings Jackson "Broadbent's Hypothesis," some to illustrate the variety of his work in the early days, but most of them for the material of interest which they contain for the medical man of to-day.

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NEUROLOGY

THE EXAMINATION OF THE HEART

St. Louis Medical Review, March 30, 1907

THE examination of the heart supposed to be the seat of disease is not a mere academic exercise, and has not for its object simply the identification of a valvular lesion or structural change, however interesting such an investigation may be. The duty before us is to form an opinion as to the exact condition of the heart, its structural integrity or damage, and functional efficiency or inadequacy, as to the effect of this condition on the prospect of life of the patient, and as to how far it is compatible with work and enjoyment.

We have to decide what restrictions on exercise are necessary, and in many cases to regulate the entire mode of life.

It is only by a thorough knowledge, not simply of the existing state of the valves and walls and cavities of the heart, but of the processes by which a given lesion has been reached, and of the tendencies, favourable or adverse, which are in operation, whether as a result of the affection of the heart, or arising out of constitutional conditions generally, that we are in a position to advise and treat the patient.

We do not, therefore, at once proceed to make out the physical signs. These have to be interpreted by symptoms, by any evidences which the patient may present of interference with the circulation, such as oedema, or breathlessness, or lividity. A murmur, or even an abnormal condition of the walls and cavities, would have a very different significance according as these symptoms were present or absent.

The pulse, again, must be interrogated, not only as affording evidence of the state of the heart, but as giving information with respect to the resistance to the onward movement of the blood in the capillaries and arterioles, and to the condition of the vascular system.

DROPSY.

Of symptoms attending heart disease, dropsy is the most characteristic. When other causes of oedema or ascites—renal disease,

disease of the liver or lungs, obstruction of veins by thrombosis or pressure, anaemia—can be excluded, dropsy is significant of back pressure in the venous system and of some serious impairment of the efficiency of the heart.

If albuminuria is present it is necessary to make out whether it is secondary to the heart affection and a simple consequence, or a complication which has supervened, or, as may be the case, a concomitant of renal disease which has actually brought on the heart disease.

Dropsy does not attend all heart disease indifferently. When it is present we expect to find either dilatation or mitral insufficiency or both.

If dropsy has supervened in the course of aortic valvular disease, whether stenosis or insufficiency, it has come about through failure of the left ventricle to cope with and compensate for it, and this ventricle will be dilated. We have aortic disease with mitral symptoms, and the prognosis is grave.

In mitral stenosis we may have no dropsy from first to last, and ascites may precede anasarca.

Dropsy does not attend fatty degeneration of the heart. A certain degree of pressure is necessary in the arterial system in order that the serum may transude through the capillary walls; this is wanting when the muscular walls of the left ventricle are fatty and when the supply of blood to the ventricle is cut off by constriction of the mitral orifice.

Breathlessness.

Another symptom is breathlessness on slight exertion. This attends all forms of heart disease. It is less marked in aortic than in mitral valvular affections, most marked, perhaps, in mitral stenosis. There is breathlessness in both dilatation of the cavities and degeneration of the walls. It would perhaps be going too far to say that when no shortness of breath follows sudden sharp exertion there can be no serious disease of the heart, but the absence of breathlessness may be allowed to discount very heavily the significance of physical signs. On the other hand, great breathlessness must be regarded as serious, even in the absence of physical signs of any kind as, for example, in patency of the foramen ovale.

It must be remembered, however, that shortness of breath attends lung affections, anaemia, and debility.

Palpitation.

Palpitation is common in heart disease, but it is also extremely common when no disease is present. The discussion of palpitation

is too large a question to be entered upon at present, but it may be said that, whatever meaning may be attached to the term, it is more frequently an effect of some outside disturbing influence than an indication of disease of the heart itself.

Irregular and Intermittent Action.

An irregular or intermittent pulse and heart beat, again, can not in itself be regarded as conclusive evidence of disease, but mitral insufficiency, dilatation, and fatty degeneration frequently give rise to irregularity in the action of the heart, and all forms of morbus cordis may be attended with faltering and irregular pulse as they near a fatal termination.

Pain.

Pain, as a symptom of disease of the heart, has to be interpreted by physical signs instead of assisting in their interpretation like breathlessness. It varies in character. If it is anginoid, the first question which will arise is, whether it is true angina, i.e. primarily cardiac, or due to reflex or other influences. If the pain habitually comes on during repose it is almost certainly an effect of some disturbance from outside. The special characteristic of true angina is that it is induced by exertion and ceases when the exertion is stopped, and this conclusion is not affected by the fact that it is more easily brought on after a meal, and that cessation of the pain is often attended by eructation. Having decided that the pain is that of angina, it still remains to ascertain how far it is an effect of valvular disease or of dilatation of the aorta or of vasomotor conditions, or of obstruction of the coronary arteries and degeneration of the heart walls.

Aching.—Other kinds of pain are described as aching, or bursting, or crushing, or heavy, or sharp; in the apex region, or across the chest, or down the sternum, or in the area of the pulmonary artery. The pain may or may not be attended with tenderness on pressure; tenderness is most common in the apex region. We have always to pay attention to complaints of pain, and sometimes it is of diagnostic significance.

The Pulse.

The pulse is of the greatest importance in the interpretation of the physical signs which are met with on examination of the heart.

In valvular affections it may be absolutely diagnostic, as in aortic incompetence, in which disease it is often of far greater prognostic significance than the diastolic murmur. The large size of the artery, the suddenness of the impact on the finger, the rapidity of

the collapse, the visibility of the beat, furnish the most trustworthy criterion of the amount of regurgitation. In aortic obstruction, when considerable, the artery is small and contracted and the wave gradual. Irregularity is most frequently associated with mitral insufficiency. The most constant feature of mitral stenosis is its smallness. The output of blood by the ventricle is insufficient adequately to distend the arterial system.

While the pulse may enable us to recognize valvular disease this is by no means the most important information which it affords. The pressure within the vessel, the character of the beat, the size of the artery, and the condition of its walls, together often furnish a criterion of the condition of the entire cardiovascular system and may throw light on the cause of structural affections of the heart and aorta, on valvular lesions, and indeed on constitutional conditions generally.

Tension.—Whatever the character of the pulse it must be borne in mind in the interpretation of the examination of the heart, but it is chiefly what is called high pulse tension which is concerned in the production of changes in the heart and great vessels. The term tension has been much criticized from various points of view, and I must explain what I mean by it. Tension, as I understand and employ the term, is produced whenever there is sufficient obstruction in the capillaries and arterioles to keep the artery full between the beats, so that it can be rolled under the fingers in a cylindrical form, even in the intervals. The actual degree of pressure within the vessel will be dependent on the vigour of the systole of the heart and the amount of blood projected into the aorta, which may vary from hour to hour. The pulse, therefore, may be either strong or weak, while having the character of tension, and may be the weakest when high tension has done its worst as regards the heart, and has given rise to dilatation, and is still doing its worst by resistance with which the worn out heart cannot deal. With tension of the pulse, as here understood, the pulse may be small when the artery is tightened up, or large when it is distended by protracted high blood pressure ; the arterial walls may be thickened from hypertrophy, with or without degeneration, or thin and inelastic. Whenever high tension is present it must in a sense direct and dominate the treatment.

Occasionally an apparent contrast is met with between the heart and the pulse, the cardiac impulse being forcible and the sounds loud, while the pulse is very weak. It is a point which demands attention. The impulse may be that of the right ventricle only, or the left ventricle from some cause or other receives and propels a small charge of blood, or the current is intercepted between

the heart and the wrist, or there is adherent pericardium and the hypertrophied heart expends its strength in dragging upon the adhesions.

It is only after the preliminary inquiries enumerated, which will suggest the special direction to be taken in the physical examination, and will interpret its revelation as we go on, that we begin the examination of the heart properly speaking, and it may be said at once that the examination is not complete unless it has been made in two positions, erect and recumbent. A point on which I place special emphasis is that whenever the examination is repeated it should be as minute and thorough as if it were being made for the first time. Disease does not stand still, and slight variations may have great significance.

When the chest is exposed we take note of the extent and character of any pulsation which may be visible in the apex region or over the right ventricle or in the epigastrium. Pulsation elsewhere would demand special attention, as it would probably mean aneurism. Very commonly no pulsation whatever is visible, or only a slight beat or tug below the ensiform. Retraction of intercostal spaces will be noted. It may be systolic or diastolic. Not uncommonly what seems to be pulsation in one or two spaces is an effect of systolic retraction, and, when the finger is applied, the impulse is felt not when the space bulges, but when it falls in. This is usually an effect of atmospheric pressure, as the volume of the heart is diminished in systole, and the heart will generally be found to be enlarged, more frequently by hypertrophy than by dilatation. Occasionally systolic retraction of spaces is due to adherent pericardium. True diastolic retraction could scarcely mean anything else. There may be systolic tucking in of the infrasternal notch, which will be attributable to the systole of the right ventricle.

The neck will be scrutinized even more closely than the cardiac area. The carotid throb of aortic regurgitation is highly characteristic. It is not simply pulsation at the root of the neck, but such as can be followed upward and seen behind the ramus of the jaw. The state of the veins will be carefully observed; for example, distended jugulars, superficial and deep, with or without jugular pulsation. This jugular pulsation is often double, auricular and ventricular, and double venous pulse may be seen just above the clavicles when the jugulars are full. Occasionally there are two distinct pulsations in the jugulars to each apex beat. Distention of one jugular only, especially if no impression is made on the full vein by a deep inspiration, will signify obstruction of the vein usually by pressure of an aneurism or growth within the chest. Pressure on the vena cava may be suspected if with bilateral fullness

of the jugulars the veins remain as full as ever when a deep inspiration is made.

Palpation and Percussion.

Palpation and percussion will be carried on simultaneously, and they constitute an exceedingly important and indeed absolutely necessary part of the examination. In most cases they enable us, with the information obtained from the pulse and from inspection, to arrive at the diagnosis and prognosis before the stethoscope is applied to the chest at all. Without palpation and percussion the sounds and murmurs heard by auscultation would afford a very imperfect idea of the condition of the heart.

Apex.—We begin, then, by endeavouring to localize the apex beat by pressing the finger into the spaces in the apex region, noting the precise point at which the thrust is most distinct and the extent over which it is felt, noting, also, how far the beat is a real thrust, or a simple shock, or a mere tap. When impulse is found at the seat of the normal apex beat we do not at once conclude that it is the apex till we have explored the entire region as far out as the midaxillary line and as high as the fourth space above and outside the mamma. The apex may be found in most unexpected situations.

Very frequently the apex beat cannot be felt at all, but we do not infer from this cardiac weakness. In a deep chested individual the heart is too remote from the surface to be felt, or the lung may overlap the heart more than usual from emphysema or from normal expansion.

Three fingers may next be pressed into the spaces to the right of the upper sternum in case the aorta can be felt.

The right hand will now be applied over the entire cardiac area, the palm over the right ventricle, the fingers over the apex region close together or spread out. Attention will again be directed to the seat and character, diffuse or defined, thrusting or knocking, and to the force of the apex beat. It is when the hand embraces the praecordium in this way that the peculiar sharp tap of mitral stenosis is best recognized and the presystolic thrill felt.

While the fingers investigate the apex beat the flat of the hand will feel the impulse of the right ventricle as a simple shock or a gentle heave. When it is forcible, the right ventricle is meeting back-pressure through the lungs and compensating by more powerful action for some affection of the left ventricle or for obstruction in the pulmonary circulation.

Palpation is not restricted to the chest. The abdomen is exam-

ined, and more particularly it must be ascertained whether the liver is enlarged. So important is this, that it may almost be said that we begin the examination of the heart by examining the liver. This organ may reach an enormous size when there is back-pressure in the systemic veins, and it may be jogged by the right ventricle, or there may be true pulsation in it of a gentle expansile character as an effect of tricuspid regurgitation. This last is better seen than felt. We press firmly on the enlarged liver and watch the hand. The gentle heave can be seen when it cannot be felt.

Percussion is of special importance when neither apex beat nor general impulse can be felt, but it always supplements palpation by showing how far the dullness extends beyond the apex beat, and by mapping out the entire left border of the heart. Attention is always especially directed to the dullness to the right of the lower sternum. Dullness here is due to the right auricle and affords an early indication of distention of the right heart. As in palpation, so in percussion, the examination extends to the abdomen. Very many cases of palpitation, irregular and intermittent action of the heart, find their explanation in stomach resonance along the fifth space or rib.

We may now pause for a moment and see how far diagnosis and prognosis can be carried before we apply the ear or stethoscope to the chest.

We shall have recognized with certainty the existence of any considerable degree of *aortic insufficiency*, and shall have estimated its prognostic import by the size of the artery, the suddenness of the beat, the abruptness of the collapse, the visibility of the radial pulse and of the carotid throb, the seat, extent, force, and character of the apex beat. We shall probably have diagnosed *aortic stenosis* from the rather small and gradual, long, and perhaps bisferiens, pulse contrasted with the powerful, defined, apex-thrust of the hypertrophied left ventricle. It is more difficult to form a judgment as to the extent of the valvular lesion when there is both obstruction and regurgitation.

We cannot be so positive in our diagnosis of *mitral insufficiency*, since the diffuse and weak apex beat to the left of the normal situation, the increased area of cardiac dullness, and the extension of dullness to the right of the lower sternum, belong to dilatation of both sides of the heart, and such dilatation may arise independently of any valvular lesion. The same may be said of irregular action of the heart, jugular pulsation and distention, and of symptoms due to venous stasis and back pressure. The pulse, however, may help us; arteriosclerosis and the remains of high tension

would point to obstruction in the peripheral circulation as a sufficient cause of dilatation; a short pulse with thin-walled vessels would suggest primary mitral disease.

Mitral stenosis is usually very definitely recognizable by the remarkable tapping character of the apex beat, not far from the normal situation. A presystolic thrill may or may not be felt, but, when present, it is unmistakable. The pulse will be small and full between the beats and will contrast with the powerful cardiac impulse resulting from hypertrophy of the right ventricle. There will be dullness to the right of the lower sternum from distention of the right auricle, probably double jugular pulsation, possibly enlarged liver and true hepatic pulsation.

Combined mitral stenosis and insufficiency present difficult problems, which can only be elucidated by auscultation.

Hypertrophy, shown by displacement downwards of the apex, with a well-defined, powerful apex thrust and general impulse, when not compensatory of aortic valvular lesions, will be evidence of old-standing high arterial tension and may be suggestive of renal disease.

Dilatation with a diffuse apex beat to the left of, and perhaps above, the normal, and more or less right ventricle impulse, with extension of dullness laterally in both directions is evidence of failure to counteract some distending influence, valvular or peripheral, or of loss of tone and vigour in the muscular walls of the heart, arising from acute febrile disease, or from debility, or from alcoholic, or toxic influences.

When no apex beat or impulse can be felt, we may exclude hypertrophy. Dilatation will be indicated by extension of the area of dullness. When the dimensions are practically normal, the diagnosis may lie between a perfectly normal heart, fatty degeneration, and mere asthenia, temporary or chronic. The distinction can be effected only by auscultation and then often with difficulty, but help will be afforded by the presence or absence of symptoms and by the pulse. A pulse of normal character in all respects will argue a normal heart, while a short unsustained pulse will suggest degeneration. A fatty heart is incapable of a sustained pulse.

Adherent pericardium may be suggested by a marked contrast between a powerful cardiac impulse and a small weak pulse, perhaps of paradoxical character, and a confident diagnosis may often be made by means of diastolic retraction of spaces or by a combination of immobility of the epigastric triangle in respiration, imperfect descent of the apex in inspiration, and inadequate shifting of the heart when the patient turns on his right side.

Auscultation.

Coming now to auscultation, we plant the stethoscope in succession at four points, the mitral, tricuspid, aortic, and pulmonic areas, at which, respectively, we listen for the left and right ventricle first sounds, the aortic and pulmonic second sounds. The right ventricle first sound is shorter than that of the left and rather louder, and the pulmonic second sound is somewhat lower in pitch and louder than the aortic. Any changes in the character or relative intensity of the different sounds must be noted. There will be prolongation of the left ventricle first sound, with lowered intensity, in hypertrophy; shortening, lowered pitch and intensification in dilatation, when also it may be heard far out to the left, corroborating indications derived from percussion. There will be reduplication of the first sound when the two ventricles are thrown out of accord by disproportionate resistance in the systemic or pulmonic circulation. Accentuation of the pulmonic and aortic second sound will indicate increased blood pressure in the pulmonary artery and aorta respectively.

There will be accentuation of the pulmonic second sound when there is obstruction in the pulmonary circulation from bronchitis, emphysema or other affection of the lungs, or from back-pressure in the pulmonary veins from mitral stenosis or insufficiency, or from dilatation of the left ventricle; of the aortic second sound, when there is high pressure in the systemic arterial system from obstruction in the arterioles and capillaries.

When the aorta itself has become dilated from protracted high arterial tension or from primary disease in its walls, the aortic second sound will not only be accentuated, but the pitch will be lowered and it may have a ringing tone. It will also be heard some distance outwards into the chest beyond the right edge of the sternum and across the manubrium.

Reduplication of the second sound, due to want of synchronism between the closure of the aortic and pulmonic valves, is much more easily induced than reduplication of the first. It may be heard on holding the breath for a few minutes or simply when a patient lies down, or for some time after effort. It is most frequently constant and habitual in cases of mitral stenosis and adherent pericardium, and is usually heard in the early stage of pericarditis.

While reduplication of the first sound is mostly heard in the apex region, and of the second at the base, it is by no means always easy to say whether the *bruit de galop*, or cantering rhythm, is due to reduplicated first or second sound. It may seem at one moment to be one, at another the other. In such a case we find some point over the heart at which we can with confidence identify and dis-

tinguish between the two sounds, and from this we work towards the apex and base.

Intervals.—Attention must be paid to the intervals as well as to the sounds. Deviations from the normal rhythm are full of significance. The short systolic space between the first and second may be prolonged so that the sounds become equidistant, like the tic-tac of a pendulum. This is incident to high arterial tension and mostly when it has given rise to dilatation of the left ventricle. Equidistance may be arrived at in another way, by shortening of the diastolic pause. It occurs when the heart is acting rapidly, as in palpitation or in tachycardia ; the sounds may now be compared to the ticking of a watch and remind one of the foetal heart sounds in utero.

A deviation from the normal of great importance is the shortening of the first pause so that the second sound comes on top of the first. This may be due to abrupt contraction of the ventricle, with diminished resistance in the peripheral circulation, or it may mean that the ventricle does not go through with its systole but is brought up short either from inherent weakness or by resistance which it cannot overcome. This approximation of the two sounds may be met with in many forms of heart failure. It may in particular give many hours' warning of fatal syncope in diphtheria or in the last stage of typhoid fever.

Perversions of the intervals, when the first sound is short, may aggravate the difficulty of saying which is first and which second. We have to try to find a point at which the distinction becomes clear. Occasionally we meet with a case in which it seems as if the sounds heard at the apex could not belong to the same heart as that which produced the sounds at the base.

Murmurs.—With regard to murmurs, it is scarcely necessary to say what murmurs attend the different valvular affections. While, however, they indicate the seat and character of the lesion, they do not give definite information as to its degree. A murmur may mean anything or nothing—in one case obstruction or insufficiency of a most serious kind ; in another a mere roughness or a trifling leakage. Everything depends on the amount of damage which the valve has sustained. This is what we have to estimate. In proportion as the valvular lesion is serious, so will be the changes in the cavities and walls of the heart, damaging or compensatory, to which it has given rise, and, when compensation is inadequate, the symptoms which betray the impaired circulatory efficiency.

We first, therefore, fall back on the symptoms, breathlessness, dropsy, etc. ; then on the indications afforded by the pulse, the vessels in the neck ; and finally, on the evidences of dilatation and

hypertrophy of the heart furnished by palpation and percussion. While, however, we rely mainly on these, the murmurs themselves may contribute to our knowledge.

We may first dismiss certain pseudo-murmurs—the cardio-respiratory murmur produced by displacement of air in overlying lung, chiefly in the neighbourhood of the apex. It is usually present only during inspiration, and is suspended when the breath is held. In some cases it is present in the erect, in others in the recumbent posture. It may be heard over a great part of either right or left chest when there are pleural adhesions, and may, on the left side, be taken for a mitral murmur conducted round to the back

Another pseudo-murmur is a systolic scratch sometimes heard over the right ventricle near the ensiform. It marks the position of a white patch of thickened pericardium occasioned by close contact of the heart with the chest wall ; not uncommonly the lower end of the sternum is depressed.

These imitation murmurs are outside the heart, but there are true cardiac murmurs which are of no consequence. The most common is the systolic murmur heard over the conus arteriosus of the pulmonary artery. It varies in intensity, even in the same case, and is usually louder, and heard over a larger area, in the recumbent than in the erect or sitting position, sometimes so far down towards the apex as to be taken for a mitral murmur. It may be very loud and harsh, especially in women. It is most common in the young, but may be present at any period of life and may be temporarily induced by exertion. It is due to contact of the conus of the right ventricle with the chest wall. Eddies in the blood and vibrations of the walls of the conus and artery are generated which are heard as a murmur. If a deep inspiration is taken and held so as to bring a cushion of lung over the heart the murmur is no longer produced.

A very remarkable loud harsh murmur due to congenital malformation of the heart, perforation of the upper part of the interventricular septum, is sometimes heard in the area of the pulmonary artery. It is continuous throughout the cardiac cycle but is intensified at the systole and may be compared to the sound of a knife-grinder's wheel intensified by the stroke of the treadle. I do not place this in the same class as the pulmonic systolic murmur just spoken of, but in none of the numerous cases which I have seen have symptoms attended it.

An inconstant systolic tricuspid murmur may be perfectly harmless.

Taking now the valvular murmurs in turn, it may be said in general terms that, if one must have a murmur, the louder it is the

better. There are exceptions, but intensity testifies at any rate to vigour of the systole.

Beginning with mitral insufficiency, the murmur which is systolic in time and is heard at or to the left of the apex affords us some help in estimating the amount of the reflux into the auricle. It may be headed by a more or less distinct first sound, which is favourable, or, on the other hand, it may entirely replace it, which is indicative of free regurgitation, or of weakness of the ventricle, or of both. In proportion as the murmur can be followed to the left of the apex round the chest and is conducted through to the left interscapular space it signifies dilatation of the left ventricle and, therefore, free regurgitation. The degree of accentuation of the pulmonic second sound will correspond with these indications and will be considerable. If a murmur is heard only over a limited area at the apex and begins with a good first sound the regurgitation is not very large, and we should expect to find little accentuation of the pulmonic second sound, very slight displacement of the apex to the left, very little extension of dullness.

A question often arises whether a systolic apex murmur is due to a valvular lesion, or simply to a want of contractile vigour in the muscular fibres surrounding the orifice. The distinction may be of great prognostic importance in the young, when the latter condition may constitute what has been called "curable mitral regurgitation." Late in life there may not be much to choose between the two. It is the presence of a distinct and perhaps loud first sound heading the murmur which distinguishes the curable, asthenic, mitral regurgitation from a true valvular defect.

A musical murmur, unless there is also a murmur of a blowing character, may be understood as showing that the reflux takes place through a narrow chink between the flaps of the valve and is therefore not free.

In mitral stenosis the information obtained by auscultation has a more direct bearing on diagnosis and prognosis than in mitral insufficiency. The presystolic murmur and thrill, the sharp tap, which represents the apex beat, and the short, sharp first sound are diagnostic of mitral stenosis. No indication of the degree of constriction of the orifice is afforded by dilatation or hypertrophy of the left ventricle. For this we look to modifications in the murmur as the constriction increases. In the first stage of mitral stenosis a short vibratory murmur, best heard to the inner side of the apex, runs up to the first sound, which is short and sharp, and is followed by an audible aortic second sound conducted to the apex. In the second stage the aortic second sound is no longer heard at the apex, and the presystolic murmur will be longer, that is, it begins

sooner. It may, indeed, come to occupy the entire diastolic interval, when the first part of it represents the suction action of the ventricle as it expands after the systole, the second part being produced by the systole of the auricle. The last part may after a time make default when the auricle is paralysed by over-distension, leaving only the murmur attending the suction action of the ventricle, which will be diastolic in rhythm. Or the entire murmur may disappear, and there may remain only the sharp apex tap, by which time a systolic tricuspid murmur will probably have been developed.

These changes in the murmur mark increase in the constriction of the orifice.

The pulmonic second sound is accentuated from the first, and it is in mitral stenosis that it reaches its greatest intensity. Reduplication of the second sound at the base is very common.

In aortic insufficiency the large collapsing pulse, the carotid throb, and the dilatation and hypertrophy of the left ventricle constitute the criterion by which we estimate the degree of regurgitation, but we also learn much from auscultation. If the murmur is long, the reflux is not rapid and the valves are offering resistance, i.e. the insufficiency is not very great; if short, the rush of blood back into the ventricle is considerable and the lesion serious. If, again, the second sound is well marked and heard in the carotids, where there is no danger of the pulmonic second sound being taken as aortic, the valves are sufficient to offer a decided check to regurgitation. The second sound may, indeed, be loud, low-pitched and ringing, in spite of the diastolic murmur, when the insufficiency is due to dilatation of the aorta, as a result of arteriosclerosis, the valves being undamaged, but no longer capable of closing the enlarged orifice. The absence of the second sound is an indication of serious damage to the valves.

The murmur indicative of aortic stenosis is systolic, but in a large proportion of the cases in which a systolic aortic murmur is present there is no obstruction whatever. The murmur may be due to roughness or irregularity at the orifice, to rigidity of one or more of the cusps, to dilatation of the aorta above the ring, or possibly to anaemia. We look to the pulse and to the existence of compensatory hypertrophy for evidence of actual stenosis. A good second sound negatives it.

A difficult problem is often presented by combined aortic stenosis and insufficiency. The two conditions neutralize each other as regards modifications of the pulse. The degree of hypertrophy and dilatation will bear witness to the existence of a valvular lesion. The aortic second sound again becomes an important criterion; both obstruction and regurgitation tend to impair and obliterate it.

THE CONDUCT OF THE HEART IN THE FACE OF DIFFICULTIES

Medical Society's Transactions, 1899

MR. PRESIDENT,—I should like the Society to understand that I am in no way responsible for the title of this communication, but that I accepted it at your dictation. There are appeals more imperative than commands. Such was yours when, as an old pupil become President of this Society, you requested me to read a paper on "The Conduct of the Heart in the Face of Difficulties." I was bound to obey, but I ventured to point out that the word "conduct" in this phrase might have two distinct meanings—the behaviour of the heart itself under difficulties, or the way in which it might be helped and guided through them by the physician, and to ask which of the two questions was to be discussed. You promptly answered "both." It is by your command, therefore, that a *double entendre* is inflicted upon the Society. In dictating the title you practically also decided the way in which the question was to be treated.

Difficulties from outside.—The difficulties which the heart has to face are sometimes of its own making, sometimes imposed upon it from outside. An over-distended stomach, for example, pushes up the diaphragm and presses upon the right ventricle; effusion into the pleural cavity, or more rarely a mediastinal tumour or aneurism, carries the heart to one side or other of the chest; or the flow of blood towards the right auricle may be impeded by pressure on the superior vena cava. Difficulties of another kind may arise from obstruction in the arterioles and capillaries—in the pulmonic circulation by emphysema or by acute affections of the lungs; in the systemic circulation by the numerous conditions which give rise to high arterial tension.

The heart adjusts itself to altered conditions in a wonderful fashion. It may be so much displaced by effusion into the left pleural cavity or by traction from a shrunken right lung that its beat is felt as far out as the right nipple, and yet we can trace very little inefficiency in the functional action. Time, however, is an element in this accommodation. The same degree of displacement occurring suddenly, as in pneumothorax, will be attended with severe dyspnoea, and great disturbance of the action of the heart

will be occasioned by sudden and capricious distension of the stomach.

The difficulties arising out of *flatulent distension of the stomach* or colon or intestinal canal generally, will require some attention, since they are the cause of most of the functional derangements to which the heart is subject, and give rise to the heart complaints which occasion in the aggregate perhaps more suffering than does actual heart disease. The heart often tolerates a considerable degree of upward pressure of the diaphragm, and it is not uncommon to meet with stomach resonance as high as the fifth space, and to find the apex beat displaced upwards and outwards to the fourth space and outside the nipple line, without conspicuous symptoms. But the heart behaves very differently in different subjects in the presence of flatulent distension of the stomach. It partakes of the general constitutional condition of the individual; in the strong, therefore, it is vigorous, in the weak it cannot be anything but weak. Moreover, the heart has very special relations with the nervous system; it reflects every emotion, beats high with courage, is palsied by fear, throbs rapidly and violently with excitement, and acts feebly under nervous depression; but it is not only through the cerebro-spinal system that the heart is influenced, it is in immediate relation with the vaso-motor nervous apparatus, and in a scarcely less degree with the sympathetic system generally. Normally, afferent impulses are constantly flowing from the viscera to the central nervous system, and by this reflex process their blood supply is regulated, and their functional activity is governed. These afferent impulses when perverted by functional derangement or disease may become serious disturbing influences.

The nervous system in a large and increasing proportion of people is unduly sensitive and excessively mobile, and the reactions to influences of every kind are exaggerated. In some a little emotional excitement gives rise to palpitation, and a piece of bad news or the bang of a door seems to stop the heart altogether. There is in such subjects no form or degree of cardiac disturbance which may not be caused by indigestion, scarcely any symptom of cardiac disease which may not be simulated. Add a touch of hysteria on the look out for symptoms and for some one to give ear to the narration of the unparalleled agonies of the sufferer, and the difficulties of the heart, and it may be added of dealing with them, are complete.

It is of course of the greatest importance that we should be able to distinguish these functional affections of the heart from troubles due to organic disease, and this is especially the case where there is severe pain in the cardiac region. The absence of physical signs

of valvular or structural change will be a help, but murmurs may be present at one or more of the orifices during palpitation when there is no valvular affection, and there may be actual mitral or tricuspid incompetence when all the symptoms are really of neurotic or dyspeptic origin.

Angina pectoris is one of the cardiac affections which may be closely simulated by the effects of dilatation or functional derangement of the stomach. The first question to be put in a case of cardiac pain of anginoid character is "As to the circumstances under which it comes on?" Whether as an effect of exertion or during repose? The earlier attacks of true angina are practically always provoked by exertion, while spurious angina is specially liable to come on during repose. It is true that angina when established may come on in the night, or may be induced by the act of undressing and the contact of cold sheets, but there will be a history of attacks during exertion. Pain and a sense of suffocation may also be brought on by the pressure of the abdominal viscera reinforcing that of a distended stomach on lying down, whether the heart is diseased or sound, and a weak heart may actually be brought to a standstill in this way. Speaking generally, angina pectoris in a woman is always spurious, and the more minute and protracted and eloquent the description of the pain the more certain may one be of the conclusion. Again, when palpitation or irregular action of the heart, or intermission of the pulse, or pain in the cardiac region, or a sense of oppression follows certain meals at a given interval, or comes on at a certain hour during the night, there need be little hesitation in attributing the disturbance, whatever it may be, to indigestion in one or other of its forms. Nightmare from indigestion is not a bad imitation of true angina. So also if any cardiac symptom or pain can be walked off, it may usually be set down as functional, and due to some outside disturbing influence or to nervous irritability. The same may generally be said of intermission of the pulse, of which the patient is conscious, and, though with less confidence, of irregularity of the heart's action—if the patient feels it the irregularity is usually temporary, and not the effect of organic disease.

In these functional affections it is not the heart which is to blame; it is more sinned against than sinning, and if its difficulties are removed there will be nothing to find fault with in its conduct. The difficulties are, as has been said, the state of the nervous system, on the one hand, and of the digestion on the other, and according as the neurotic or the dyspeptic element predominates will be the treatment required. No details need be entered into, but one observation may be made. Patients suffering from these functional

derangements of the heart usually make them a pretext for avoiding exercise, and often for taking stimulants or drugs, whereas exercise and fresh air are what he or she most needs. The best way to prevent the expenditure of superfluous energy on the part of the heart in the form of palpitation is to give it a fair amount of legitimate physiological work to do; and to relieve one attack of palpitation or faintness by alcohol is to invite another, while the terrible danger of drifting into alcoholism is incurred.

High arterial tension.—One of the most common difficulties with which the heart has to contend is high arterial tension, or rather the obstruction to the onward movement of the blood in the capillaries and arterioles which is the cause of the high pressure in the arteries. While dyspeptic troubles and other reflex sources of irritation give rise merely to functional affections of the heart, high arterial tension when persistent is a frequent cause of actual disease. The resistance in the peripheral circulation has to be overcome and the heart rises to the occasion. It puts forth the increased energy required, and in doing so becomes hypertrophied. Hypertrophy is not disease, though sometimes the heaving impulse and powerful throb of the apex are complained of by the patient and looked upon with suspicion by the medical man, but the development of additional muscular fibre is accompanied by the development of increased connective tissue, and when in the decline of life the nutrition of the more highly organized structures is no longer vigorous, the fibroid element may gradually predominate over the muscular, or fatty degeneration may take place.

But the valves may suffer before the muscular walls. Where greater force is required to propel the blood into the aorta there is greater strain upon the mitral valves during systole, and a more violent recoil upon the semilunar aortic valves during diastole. This gives rise to chronic inflammation of the valves, with thickening and contraction, and, in the long run, insufficiency.

It was necessary to mention high arterial tension on account of its frequency and importance as a source of cardiac difficulty, but I have dealt with it so often and so recently that I will forbear from further dwelling upon it on the present occasion, only remarking that the recognition of unduly high pressure in the arteries affords one of the most valuable indications for treatment in a great variety of conditions.

I am afraid it often escapes recognition, and sometimes digitalis is given for the relief of the cardiac discomfort which may attend it. This is like knocking the head against a stone wall, for digitalis not only acts on the heart, but tightens up the vessels, and so increases the obstruction, already too great.

Valvular disease.—Coming now to the serious difficulties to which the heart is exposed by reason of damage to one or other of its valves, we discover, say, a systolic murmur at the apex or at the right second intercostal space, indicative of leakage of the mitral valve or of interference with the blood current at the aortic orifice. What are we to do? Frighten the patient out of his life or out of his peace of mind? Condemn him at once to live on one floor, and forbid him exercise and excitement and all that makes life tolerable, and give digitalis? Certainly not. Or, shall we ignore the murmur on the chance that it may not be serious, which is a not uncommon proceeding when a medical man has predicted sudden death once or twice, and found the patient to go on living for 10 or 20 years? This would be equally unreasonable.

The first thing to be done is to ascertain what the murmur really means; whether, when it is mitral, there is much or little regurgitation, or, if aortic, whether it signifies mere roughness or actual constriction. Numerous considerations enter into the determination of these questions, of which we need specify only those arising out of the conduct of the heart. If, in the case of mitral incompetence, there is any considerable reflux into the left auricle, the first effect will be damming back of the blood entering it by the pulmonary veins, and the obstruction thus created will make itself felt in the pulmonary artery, raising the blood pressure within it. There is no branch of the pulmonary artery on which we can place our finger or a sphygmograph, but the high pressure is at once accused by accentuation of the pulmonic second sound. If the circulation is to be maintained under these circumstances, something must be done to overcome the obstruction in the pulmonary circulation and neutralize the mitral reflux. This can only be by increase in the capacity and strength of the right ventricle. The right ventricle accordingly becomes dilated and hypertrophied, and the dilatation and hypertrophy which we call compensatory become for us the measure of the regurgitation. This is the conduct of the heart in the face of this particular difficulty, and we learn from the amount of compensatory change required to neutralize the effects of the valvular lesion whether the lesion is severe or slight; our conduct, then, will be guided by the degree of efficiency of the compensation. When there is no appreciable hypertrophy of the right ventricle or marked accentuation of the pulmonic second sound, and the patient has no heart-symptoms, the murmur means nothing, and there is no need to interfere in any way with the patient's mode of life, even if this include hunting, or climbing, or swimming, or cricket. I should draw the line at football or training for races of any kind.

If with marked hypertrophy and dilatation there is still no breathlessness on ordinary exertion, or other symptom of disturbed circulation, the regurgitation is considerable, but it is neutralized by the compensatory changes. We are not called upon to do anything, but the patient must be warned that the compensation may easily be broken down, and that a single imprudent act of violent or sustained exertion may do irreparable injury.

Cardiac symptoms, such as breathlessness on slight provocation, show that the compensation is inadequate, and it is only by great carefulness that the serious effects of the valvular lesion can be put off. Let us suppose that we have the heart landed in extreme difficulties from incompetence of the mitral valves, the liver enlarged till its lower border crosses the abdomen at the level of the umbilicus, the veins of the neck distended and pulsating, the face and lips livid, the lungs congested, the legs dropsical, the urine scanty, turbid, and albuminous, the patient gasping for breath and unable to lie down. It is in such a case that digitalis and the like remedies find their opportunity. But first the right side of the heart must be relieved from the over-distension which is paralysing its efforts. Unless this is done, the digitalis may simply help the straining ventricle in the work of self-destruction. The nearest approach to a modern therapeutic miracle is seen on bleeding in a good case of this kind. When I say a good case I mean one in which the onset of the severe symptoms has been sudden, under the influence of some adequate exciting cause, such as over-exertion or chill, in a fairly robust subject with a powerful right ventricle. The venesection must be followed up by a good calomel purge—2 or 3 grains of calomel, with, say, 5 grains of colocynth and hyoscyamus—and perhaps a dose of white mixture. Bleeding, however, is too heroic a method for these degenerate days, and it is not always easy to say whether it is really demanded. A good alternative is six, eight, or a dozen leeches over the enlarged liver, followed up, of course, by the calomel purge. In less severe cases we may content ourselves with the mercurial aperient alone.

The right heart having been relieved, digitalis may be given with excellent effect in different combinations, according to the condition—with nux vomica and ammonia, and perhaps ether, or with acetate of iron and potash. If the oedema is considerable, the fluid should be drained off by Southey's tubes, and any pleural effusion should be withdrawn by aspiration at an early stage.

Mitral incompetence will serve as an illustration of the difficulties imposed upon the heart by disease of the valves. They differ in the different valvular affections, and the heart responds in a special way for each one. It is unnecessary to go into details with regard to

all of them, and the time at my disposal would be quite insufficient. The principle which I wish to emphasize is, that *when the heart is in difficulties we can generally give more relief, indirectly, by removing these difficulties, than directly, by aiding it to overcome them.* This is the case; whether the disturbing influence is external to the heart—as, for example, a dilated stomach, or distended colon, or resistance in the peripheral circulation—or is a secondary effect of disease of the heart itself, as illustrated by over-distension of the right ventricle. To take another instance, if the heart is in a state of fatty degeneration, it is useless to give cardiac tonics, its work must rather be diminished by keeping down the arterial tension, and a fatal issue may be for a time averted by preventing distension or dilatation of the stomach. Such illustrations might be multiplied indefinitely.

When, therefore, we are considering the treatment of cardiac disease, the first question to engage the attention is how we can relieve the labouring or harassed heart by the removal of some condition which is causing or aggravating the difficulties with which it is already contending. In doing this we often put an end to the symptoms which have given rise to suffering and anxiety, and in all cases we make the action of digitalis or other cardiac tonics more efficacious.

ADHERENT PERICARDIUM

Medical Society's Transactions, 1898

Mr. President,—I am greatly honoured by your invitation to open a discussion before the Medical Society on "Adherent Pericardium." I take it that what is expected of me is not a full and complete consideration of adhesion of the two layers of the pericardium, but a brief account of my observations and reflections which may invite comment and criticism and contributions from the experience of other physicians.

I think I am right in believing that adherent pericardium often escapes recognition, and that a definite diagnosis of this affection is not often made, or perhaps attempted, and I am sure I am right in saying that the diagnosis is often extremely difficult, and sometimes that it is practically impossible to arrive at anything more than a conjecture of its existence. Partial adhesions may give rise to neither symptoms nor physical signs, and even adhesions involving the entire surface of the heart may be attended with little or no interference with its functional efficiency, and therefore with no appreciable hypertrophy, or dilatation, or disturbance of the circulation calling attention to the heart, provided there is no outside adherence of the pericardium to the chest-wall. It might, perhaps, be going too far to say that when adherent pericardium has given rise to neither symptoms nor recognizable change in the dimensions or form of the heart it can have no importance whatever, for while it may not sensibly impede the action of the heart during ordinary exercise, or even occasion unusual shortness of breath on exertion, it might hamper the right auricle and ventricle in case of bronchitis, or interfere with the compensatory hypertrophy of the left ventricle in case of renal disease or other condition attended with high arterial tension. Still more might it aggravate the effects of valvular disease; or the degenerative changes of old age in the muscular walls of the heart may be antedated, or fibroid infiltration may invade their substance from the fibrous tissue which binds the two pericardial surfaces together.

Symptoms.—The symptoms to which adhesion of the pericardium gives rise have nothing characteristic about them. Breathlessness and a sense of oppression in the chest, palpitation, frequency or infrequency, intermittence or irregularity of the pulse, praecordial pain, sometimes severe enough to be worthy of the name "anginoid," have all been justly attributed to adherent pericardium, but each may

have various other causes. It is only when other causes have been excluded that we can with confidence look upon such symptoms as due to pericardial adhesion. Very much the same may be said with regard to dropsy and sudden death; adherent pericardium may occasionally be responsible for either of these consequences of heart disease, but this is exceedingly rare in comparison with valvular and structural affections. Sometimes, however, the absence of stasis in the pulmonary circulation when general dropsy is present from systemic venous obstruction may show that the venous obstruction has its origin in the right side of the heart, which may be hampered by pericardial adhesions, and not in the left ventricle or lungs.

I am tempted to mention one case in which such symptoms were of themselves almost sufficient to establish a diagnosis of adherent pericardium. In a young man, otherwise strong and healthy, and with no renal or valvular disease, oedema of the lower extremities came on rather rapidly, and two or three times subsided as rapidly when he lay up for a few days. It then became persistent in spite of rest, and very soon enormous. There was never at any time evidence of back pressure through the lungs, or of obstruction in the veins of the neck, and it was remarkable how little the patient suffered. The oedema was relieved time after time by Southey's tubes, but at length death supervened from cellulitis, when it was found that the pericardium was universally adherent, but also that the right auricle was practically obliterated by the adhesion surrounding it, so that the superior vena cava was directly over the inferior. Having the advantage of gravity, its stream had dammed back the ascending current in the inferior cava, and this vein was enormously dilated. An explanation was thus afforded of the absence of stasis in the lungs, and of enlargement of the jugulars.

Valvular disease with adherent pericardium.—A very common problem presented for solution is whether in a given case of valvular disease there is not also adherent pericardium. The different forms of disease of the valves are attended each with its own characteristic effect on the cavities and walls of the heart, by means of which compensation is more or less perfectly attained, and the degree and kind of dilatation or hypertrophy become a criterion by means of which we estimate the amount of obstruction or regurgitation. But this criterion sometimes fails us. For example, a well-marked collapsing pulse, carotid throb, capillary pulsation, and absence of the aortic second sound indicate very considerable insufficiency of the aortic valves. The patient is of an age when compensation ought to be promptly established, and we expect to find a powerful diffuse apex thrust in the sixth space

or lower, extending probably outside the vertical nipple line. But we are disappointed, the dilatation is comparatively slight, the hypertrophy poorly developed, and the patient suffers from inordinate shortness of breath. A presumption arises, to be verified or not by careful investigation, that the development of compensatory changes has been prevented by adherent pericardium. Or the very contrary may be present, very little regurgitation may be indicated. Although there is a double murmur, the collapse of the radial artery and the carotid throb are little marked, and the aortic second sound is distinctly heard in the neck, and yet the heart is obviously enlarged and labouring. It has something to contend with besides the regurgitation—possibly adherent pericardium.

Similar illustrations are furnished even more frequently by mitral disease, since the work of compensation falls upon the right ventricle and this ventricle is more hampered by adhesions than the left, both because it is thin-walled and not competent to contend with the obstacle to its complete contraction and because, forming, as it does, the greater part of the surface of the heart, it presents a larger area for adhesion. Being thin-walled, again, a larger proportion of its thickness is implicated and damaged by any myocarditis which may have accompanied the pericarditis or by fibroid penetration from the surface.

Diagnosis.—The diagnosis is arrived at by means of physical signs, and there is no better field for minute observation and careful discrimination. We shall better know what to look for and what value to attach to any deviations from the normal which we may discover if we consider the conditions present.

In the normal state the heart glides over the central tendon of the diaphragm on which it rests, both with its own systole and diastole and with the respiratory ascent and descent of the diaphragm. When adhesion takes place all shifting and gliding must cease; a given area of the surface of the heart (which will correspond very nearly on the posterior inferior aspect with Sibson's fixed point in the interventricular septum in front) is bound to a definite part of the tendinous expansion. While the heart is thus fixed the respiratory excursion of the central tendon must also be restricted, since the heart is adherent to the pericardium as well as to the diaphragm, and the fibrous connexions of the former do not allow of its free movement downwards. Now, the triangular space between the diverging costal cartilages is closely associated with the central tendon of the diaphragm, and it is here that we look for evidences of the adherence of the heart thereto. Visible or palpable pulsation does not help us, for we may have either epigastric protrusion or tug in normal conditions. Much more significant is

the complete arrest of the slight respiratory movements of this part of the abdominal wall. It has been my practice for more than 20 years to note carefully the indications of adhesion left by general pericarditis in every case which has come under my observation, and this arrest has never been wanting. It cannot be said, however, that immobility of the epigastric triangle necessarily implies adherent pericardium.

Other evidences of fixation of the heart are imperfect descent of the apex-beat during inspiration and inadequate shifting of the cardiac impulse when the patient lies first on one side and then on the other, more particularly when he turns upon his right side. As a rule, in normal conditions, the apex, when its beat is recognizable, disappears from the fifth space and is felt in the sixth on a deep breath being taken and held, and moves for an inch or so towards the middle line, descending also somewhat, when the patient lies over on the right side. Similar shifting of the apex-beat and of its maximum impulse may be observed even when the heart is dilated and hypertrophied in consequence of valvular disease. Distinct evidence of free mobility obtained in this way would exclude pericardial adhesion, and a marked failure to respond to the test would raise a strong presumption of its existence. But nothing must be accepted as absolute in clinical investigation, and the exercise of judgment will be called for in estimating the significance and value of the results obtained. For example, whether the pericardium is adherent or not a deep inspiration may bring the lung over the heart and the apex-beat may be altogether obliterated, its disappearance from the fifth or other space, therefore, is not conclusive of mobility. Again, when the patient is turned over on his right side a transference of the seat of maximum impulse may simply mean that another part of the heart has been brought in contact with the chest wall, and not that the apex-beat has shifted. Not unfrequently, however, from one cause or another, no apex-beat or impulse of any kind can be felt, so that palpation affords no assistance whatever and percussion cannot be relied on to furnish the kind of evidence required to distinguish between fixation and mobility of the heart.

Indications may be furnished by dilatation and hypertrophy, which may sometimes be quite conclusive when there is no valvular disease. The dullness in a characteristic case usually begins in the third space, and the apex-beat will be at the nipple level or even higher and outside the mamma. It will probably not shift with a deep breath, but it may be obscured. The transverse position of the heart and the fixation of the apex above its normal point of contact with the chest wall I formerly looked upon as a conse-

quence of effusion which had carried the apex upwards and left it adherent there. I am now convinced that the cause is not effusion but dilatation. There may be conspicuous systolic falling in of the fourth and fifth space, and, although this may be due to atmospheric pressure when the heart is very greatly enlarged from any cause, it may be a true retraction or tugging. Diastolic tugging may sometimes be felt when the hand is applied over the region of the apex. There is not merely a subsidence of the push but a sharp shock as if the chest wall were dragged upon from within, which is quite different from what is felt even after the most powerful thrust which is given by the dilated and hypertrophied heart of aortic regurgitation.

A systolic tug of the left false ribs posteriorly communicated by the diaphragm may be conspicuous. The recoil from the drag may be so distinct as to look and feel to the hand like pulsation, and in the first case in which I observed it, now more than 20 years since—a case of left empyema—it was taken for pulsation, and it was supposed that a pulsating tumour of some kind underlay the empyema. A post-mortem examination showed that the cause was adherent pericardium. I have often seen this tugging since, and in some cases it can be made to affect the right false ribs by causing the patient in the sitting posture to lean over to the left so as to throw the drag of the heart upon the right half of the diaphragm. It must be added that this indication is not infallible, as the tugging has been observed when the heart was hypertrophied without adhesions.

The pulsus paradoxus has been observed in adherent pericardium, and an effect on the veins in the neck has been described, but I have not found either of these indications helpful. In several cases I have seen a very pretty confirmation of the diagnosis in an apparent pulsation of a small tortuous vein on the front of the chest penetrating the third space near the sternum. On careful examination the vein was seen not to fill by reflux during systole but to be emptied by a sharp suction action. It was inferred and verified post-mortem that the internal mammary vein into which the surface vein opens was compressed during diastole and that its walls were dragged apart by the systole.

Auscultation does not afford much assistance in the diagnosis of adherent pericardium. Reduplication of the second sound is very common when no other indication, except perhaps immobility of the epigastrium, is present, but it is so easily induced that no definite conclusion can be based upon it, unless, perhaps, when it is constant in all positions of the body and under varying conditions of the circulation.

CLINICAL ILLUSTRATIONS OF ANGINA PECTORIS AND PSEUD-ANGINA

An Address delivered before the Brighton and Sussex Medico-Chirurgical Society

Lancet, 1905, Vol. I

The Attacks.—It is not necessary that I should describe angina pectoris in all its varieties. We understand by it pain in the cardiac region, chiefly behind the sternum, with radiation, most frequently down the inner aspect of the left arm to the elbow, or to the hand, especially the two ulnar fingers, or down both arms, or to the shoulder, neck, or face. Or it may begin in some part of the arm, as, for example, by painful tight feeling round the wrist, extending up the limb to the chest. The pain may vary in character and intensity, and may be described as crushing by a gigantic weight, or as if the chest were held in a vice, or as tearing, or burning. All sufferers agree that there is no other pain like it, and it is usually accompanied by a sense of impending death. The distinctive character of true angina is that it is induced by exertion, by walking upstairs or up an incline, or against the wind, more easily soon after food (after breakfast is a favourite time in early cases), or when the air is cold on first going out. Or an attack may come on when the patient goes to bed, after perhaps walking upstairs, the exertion of undressing, assuming the horizontal position, and the contact of cold sheets. Next to exertion in provoking an attack comes excitement, and, in advanced cases, the movement of a limb, a breath of air, the slamming of a door, the announcement of a visitor, may be sufficient to precipitate an attack.

The effect on the circulation common to all these exciting causes is increase of the blood pressure in the arterial system, and this must be regarded as the determining antecedent. The attacks are usually brief, the sufferer, indeed, will often say that if they had lasted another minute he must have died, he stands still, or suspends any exertion in which he is engaged, and may not even dare to breathe. With the cessation of the exer-

tion the paroxysm subsides, or speedy relief may be obtained by inhaling amyl nitrite or taking nitro-glycerine. The patient may then be able to resume his walk and to go on for some time without provoking another attack. Sooner or later sudden death usually occurs during, or at the onset of, a paroxysm. While the attacks are, as a rule, soon over, the pain may last for hours. In one case to which I was called the patient had spent an entire night sitting in a chair, not daring to move, the face bathed in cold perspiration and having an agonized expression. He made a temporary recovery. In another, in which I made a post-mortem examination and found the heart in an advanced stage of fatty degeneration with calcified coronary arteries, death took place after hours of terrible suffering in the night.

During the paroxysms the one condition of the circulation which seems to be always present is *contraction of the peripheral arterioles.* It was the recognition of this fact and of its significance which formed the basis of Sir Lauder Brunton's employment of amyl nitrite and nitro-glycerine, which have been of such value in the treatment of angina. I have sometimes been tempted to think that in certain cases the initial event is that the blood supply from the heart fails and the arteries contract down upon the empty lumen as they do post-mortem. The pulse may remain steady or become irregular; it is rarely much accelerated but may be slow and hesitant. The face may be flushed or pale, or keep its natural colour. The most constant post-mortem condition found has been more or less occlusion of the coronary arteries from a calcareous or atheromatous or sclerosed condition of these vessels with perhaps thrombosis, or from disease of the aorta narrowing their orifices. As a consequence of the obstruction to the blood supply, general or local degeneration, fatty or, more frequently, fibroid, of the walls of the heart is usually produced, though death may occur before even microscopic changes in the muscular striae can be recognized. A noteworthy fact is that the cavities are almost always found to be empty after death.

Cause of the pain.—The theories, as they may be called, or explanations advanced as to the cause of the pain are very many in number. Huchard enumerates no less than eighty of these hypotheses. I shall consider them simply in so far as they concern prognosis and treatment, and from this point of view only two demand serious attention. Huchard regards the attacks as consisting essentially of what may be called cramp of the cardiac muscle due to ischaemia or privation of blood produced primarily by disease of the coronary arteries or occlusion of their mouths, possibly intensified by spasmodic contraction of these vessels.

For Huchard there is only one true angina pectoris, the coronary, and his discussion of the entire question is one of the most interesting parts of his monumental work. An alternative view is that the pain is due to overstrain of the heart in the endeavour to overcome resistance in the peripheral circulation to which it is no longer equal, or to distensive stress of the aorta. The incompetence of the heart may be the result of inherent weakness from degeneration of its muscular walls, or may be only relative to great increase of the resistance in the arterioles and capillaries, or may be attributable to a combination of the two. Some degeneration or deterioration of the heart walls must, however, be an indispensable, and, indeed, the special factor in the causation, or angina would be much more common in chronic Bright's disease in which arterial tension reaches its maximum. The inadequate blood supply, again, which gives rise to the degeneration may also affect the nutrition of the cardiac ganglia and nerves and in this way predispose to pain. The conclusion that the condition of the heart walls must be the dominant factor in angina seems to me to be confirmed by two cases which have come under my observation, in which, after plague, the heart was left in a state of extreme asthenia, and pain of an anginoid character was experienced on every slight exertion.

Pseud-Angina.—Before entering upon the consideration of the different forms of true angina we may speak of false angina. To establish this distinction will be a matter of vital importance, as it may enable us to lift the burden of constant apprehension from the minds and lives of patients and to restore a family to tranquillity and happiness. We may dismiss the hysterical person, male or female, who comes with a diagnosis of angina pectoris ready-made and cannot find terms adequately to describe his or her complicated agonies, who is ready to adopt any suggestion we may make as to the seat and character of the pain and as to the direction and extent of its radiation. The attacks may come on at any convenient time and may last indefinitely, but are not allowed to interfere with pleasant engagements. These patients do not wish their friends to be undeceived as to the character of the attacks.

Paroxysmal pain in the cardiac region may be due to a great variety of conditions; it would be waste of time even to enumerate them. Speaking generally, in all cases in which the attacks come on chiefly during repose, when perhaps the patient is sitting reading or writing, however severe and however closely they may appear to conform in the description given of them to true angina, the presumption is that they are spurious, unless they have been led up to by the frequent occurrence of pain induced by exertion. The

presumption is increased if there are tumultuous action of the heart and irregularity or intermission of the pulse, of which the patient is conscious, particularly if the pain lasts for some time. The same may be said of cardiac pain occurring during the night, if there is no history of the patient having also been brought up short when walking by pain in the chest. If the nocturnal attacks recur, and particularly if they observe a sort of punctuality in their recurrence, it may be concluded that they are of gastric origin. We shall, however, be cautious in coming to this conclusion and in acquitting them of all serious significance if there is valvular disease of the heart or considerable dilatation. The cases which most closely simulate true angina are those in which there is dilatation of the stomach and when, with a dilated stomach, there is high arterial tension, which is so very frequently associated with angina, we may have attacks which can scarcely be distinguished from those of true angina. I have chosen three of these as illustrations. In all of them a diagnosis of angina had been made.

CASE 1. *Pseud-angina.*—A retired officer, aged 70 years, consulted me in October, 1895. He said he had suffered from heartburn for 30 years, and had had four attacks of influenza which had left his heart weak, so that after a walk of a quarter of a mile he was pulled up by pain in the centre of the chest which radiated down to the hands on both sides. During the attack his breathing was embarrassed and there was palpitation of the heart. He often had to lie flat on the floor. He was also liable to attacks which came on at 3 a.m. in which there was a sense of suffocation as well as severe pain and violent palpitation. He took carbonate of sodium and magnesium, brought up gas, and was then able to sleep again. The pulse was large and very tense. The cardiac impulse was weak and the apex beat could not be defined. The sounds were normal. The abdomen was very prominent; the stomach resonance was extensive, coming up to the fifth space. Voluminous splashing could be elicited. The diurnal attacks were extremely like those of true angina and they were induced by slight exertion. I have, however, never known a patient to lie flat in angina and it is very unusual for him to complain of palpitation. The attacks coming on punctually at 3 a.m. were certainly attributable to the dilated stomach, and it seemed possible that this played an important part in the production of the anginoid paroxysms which followed slight exertion. He was dieted, was given pilula hydrargyri and ipecac. and rhubarb, with the idea mainly of reducing the pulse tension, and bicarbonate and sulphocarbolate of sodium with ammonia, gentian, and peppermint for the relief of the distension of the stomach. Ten days later he had only had one attack

during which the pulse was irregular—hurried and stopping in turn. A serious indiscretion in diet was followed by distressing symptoms of a mixed character, but in December he was apparently well in all respects. I have met him since in society, sometimes very late at night. He has always said that he was perfectly well. Since the above was written he has called upon me looking the picture of health, and enjoying life in all its aspects notwithstanding his 80 years.

CASE 2. *Vaso-motor pseud-angina.*—A man of spare habit had suffered from some affection of the heart for several years. It was said to be angina, and he had twice undergone a Nauheim course. The pain was brought on by exertion, especially after food; its seat was across the upper part of the chest and down the left arm to the elbow; it was not very severe and the liability to paroxysms varied greatly. When I saw him on November 9, 1903, the pulse was somewhat tense; the dimensions and sounds of the heart were normal, but a curious dry vibratory friction was heard over the apex, especially during expiration. It resembled the sound produced by a wet finger on a pane of glass, and was exaggerated when he lay down. He had been taking iodide of potassium, and I attributed the friction to deficient moisture of the pericardial surfaces due to the iodide. The stomach resonance was extensive and the upper line was unduly high. Splashing was very distinct and voluminous; there was much dreaming and he awoke early. The tongue had a slight central fur. The treatment was suggested by the condition of the stomach and was practically such as that described. The patient was very soon able to walk uphill between 12 and 1, when the stomach was comparatively empty, but still had pain on exercise after food. On November 30 the pulse was soft and elastic, the apex-beat was felt in the normal situation, and the heart sounds and intervals were normal. He thought a tonic pill which I had prescribed did not suit him, and took again a mixture containing iodide. When next seen, on December 14, a creaking pleural friction was heard over the lower part of both lungs. The dilatation of the stomach persisted and much splashing was elicited by succussion. Sodium sulphocarbolate and carbonate were now given with ammonia and gentian three times a day. With the suspension of the iodide the pleural and pericardial friction disappeared. There was distinct general improvement but with great fluctuations, which were usually traceable to errors in diet. I found, for instance, that while drinking little with his meals, in accordance with my instructions, he took a breakfast-cup of weak tea immediately after lunch. He was also eating stewed prunes and Normandy

pippins after telling me that apples in any form disagreed with him. The condition of the stomach was not much modified, and it was difficult, as the patient was only seen at uncertain intervals, to control the eccentricities in the way of food. He was therefore taught to wash out the stomach and for six weeks did this every night. I did not see him for three months, when he could take a good deal of exercise without pain and had resumed his morning ride. When last seen, on January 25, 1905, he had remained well, though the washing out of the stomach had been given up for about six months. On the previous Sunday he had walked two miles to church and had read the lessons. He has since had occasion to write on other matters and spoke of his restoration to health.

CASE 3. *Pseud-angina.*—A man, aged about 60 years, consulted me in September, 1903, on account of pain across the chest to the left shoulder and down the arm, coming on after exertion but also at other times. He was conscious of beating of the heart and said he heard it. The pain had been pronounced to be angina, and work and exercise had been forbidden. He suffered also from giddy feelings as if tipsy. The pulse was rather tense; the heart sounds and intervals were normal, the aortic second sound being slightly accentuated. The stomach resonance was found to be extensive and its upper limit high in the chest. The diet was regulated. A mild mercurial aperient was given twice a week and measures were taken for the reduction of the distention and dilatation of the stomach. The restrictions on exercise and professional duties were gradually relaxed and removed. Recurrences of some or other of his symptoms brought this patient to me from time to time. The pulse after the first visit was of normal tension; the apex-beat was normal in character and in situation; the aortic second sound was scarcely accentuated. The dilated condition of the stomach persisted more or less, and it was found necessary to have repeated recourse to the treatment for dilatation of the stomach which I have described. I have seen him within the last few days. His duties had involved frequent journeys and much and varied work. He had shot well through the winter and walked 11 miles with enjoyment. He expressed himself as feeling perfectly well and capable of any amount of exercise, always feeling the better for it. He had pain in the cardiac region from time to time, but it had no relation to exertion and could usually be traced to irregular meals or unsuitable food.

True Angina.—Coming now to true angina, the diagnostic distinction is that the attacks of pain are brought on by exertion. The first paroxysm may come on during some special effort and may be very severe. More commonly the early attacks are

slight. Angina, answering to this criterion, is met with clinically in association with aortic valvular disease, or with aortitis, or with dilatation of the aorta, general or aneurismal, or with atheromatous disease of the aorta without obvious dilatation, or with fatty degeneration of the heart. These may be represented as different stages or different effects of general disease of the arterial system and all of them, except perhaps acute aortitis, are very common without angina. Angina is established when the coronary arteries are implicated. Disease of the mitral valve does not give rise to angina; the supervention of mitral regurgitation may, indeed, greatly diminish the liability to attacks in a case of angina. This may perhaps be because breathlessness on slight exertion anticipates the paroxysm of pain; the course of the disease may be entirely changed. In connexion with valvular affections it is only in aortic disease that angina is met with and then not usually when the insufficiency or obstruction is the result of rheumatic valvulitis. Very frequently a systolic murmur in a case of angina does not indicate real obstruction and, with a diastolic murmur, there may be extremely little regurgitation. When this is the case the valvular affection, as such, is of no importance in itself; it is simply an effect of roughness or rigidity of the ring and cusps or of dilatation of the orifice and of the root of the aorta, due to atheromatous disease in the immediate neighbourhood of the coronary arteries which has narrowed their mouths or invaded their walls. The valvular affection and the dilated and atheromatous condition of the aorta are themselves effects of old-standing high arterial tension, and the aortic second sound will be loud and ringing even when there is a double murmur, or the changes in the aorta and valves and blocking of the coronary arteries may be due to syphilitic disease of the arterial system, which is not necessarily attended with high blood pressure.

The atheromatous changes in the aorta, while ultimately exhibiting extreme forms of degeneration such as calcareous patches, begin originally as inflammatory exudations. The inflammation, however, is so insidious and chronic that no clinical evidence of its progress is recognizable, except that its presence may be inferred when physical signs of dilatation of the ascending aorta or of the arch can be made out. Occasionally subacute aortitis reveals itself by symptoms.

Case 4. *Angina from subacute aortitis.*—In November, 1899, a man, aged 50 years, who had previously been under my care for various ailments, came to consult me, looking ill and complaining of pain in the chest. He had a spasmodic cough and his voice was cracked; the left pulse was smaller than

the right. These symptoms were highly suggestive of aneurism, but no confirmatory physical signs were obtainable. A loud systolic aortic murmur, not previously present, was heard over the entire heart. The maximum intensity was at the right second space, but it was reinforced along the course of the aorta. The aortic second sound was not accentuated. This patient remained under observation for some months. He refused to believe that there was anything serious the matter with him and could not be persuaded to rest. The course of the case was extremely erratic. The pain in the chest varied in degree but could never be called severely anginoid. From time to time he looked fagged and worn, and his eyes were sunken. His nights were often bad and disturbed by the beating of the heart. The pulse was never tense and was usually below the normal frequency (60–54), sometimes small and weak, at others large and lax. The difference between the right and left radials did not last. Soon after I first saw him a soft diastolic murmur was developed. It was sometimes audible, at others not, but at length it disappeared. All symptoms subsided in about six months, but he was left with a loud systolic murmur and a sharp second sound suggestive of thin parchment-like aortic walls. The patient is still actively engaged in business. There can be no doubt that there was inflammation along the whole course of the arch of the aorta. The orifice of the left subclavian was at first apparently narrowed by swelling of the tunica intima. Fortunately the coronaries were not implicated.

In *acute aortitis*, which is very rare, there may be frequent anginoid attacks while the patient is lying in bed without any apparent exciting cause if the mouths of the coronary arteries are blocked by the swelling of the internal tunic of the aorta.

In a large proportion of the cases of angina the condition known as *arterio-sclerosis* is present in a marked degree. The radials are not only full between the beats of the pulse but the coats are thickened and firm, the vessels are tortuous and can be rolled under the finger and followed up the forearm. The radials and the brachials at the bend of the elbow can often be seen to be thrown into curves with each pulsation. The aorta is dilated. It is not always easy to make this out by percussion, but it is indicated very distinctly by the loud, low-pitched, sometimes ringing aortic second sound heard for a considerable distance outside the right border of the sternum and across the manubrium. In some cases the maximum intensity is shifted from the second to the third space by elongation of the ascending aorta carrying the base of the heart downwards. Occasionally a slight tracheal tug can be felt. The angina, however,

does not stand in any direct relation with the degree of dilatation of the aorta, but is dependent on interference with the coronary circulation. Angina may supervene in a comparatively early stage of the general arterial disease before the radials are much thickened or distinctly tortuous, and before the aorta has become dilated, if the coronaries happen to be involved in a swollen patch of exudation from the first.

In syphilitic disease of the arterial system, while there may be valvular lesions, dilatation of the aorta, or aneurism, the blood pressure may not be high. It is the part played by high blood pressure or high arterial tension in angina which affords us an opening for treatment which may alleviate and sometimes cure. The cases in which the prognosis is most unfavourable and in which we can do least for the relief of the patient are those in which the physical signs are negative. This is particularly the case in fatty degeneration of the heart. In a less degree the remark is applicable to the cases in which, with angina, there are little thickening of the radials and little dilatation of the aorta in consequence of premature implication of the coronary arteries.

Treatment.—The treatment of angina, then, resolves itself into the treatment of high arterial tension. The resistance in the peripheral circulation which gives rise to this I place primarily in the capillaries, and it is attributable to the presence in the blood of impurities which provoke the resistance. The object before us, then, is to prevent the formation and to promote the elimination of these toxins. We simplify the diet, reducing the amount of animal food, and especially of the richer meats, bearing in mind also the fact, pointed out by Dr. G. Oliver, that boiled meat raises the arterial tension much less than roast meat. Meat extracts of all kinds will be forbidden. It may be worth while in some cases to order a strict milk diet and to enforce absolute rest. The patient must, of course, be placed under the best obtainable hygienic conditions as regards air and climate, and should take daily such exercise as he is capable of without bringing on pain. Exercise may be supplemented by judicious massage and resisted movements.

By way of promoting elimination a tumbler of water should be drunk night and morning, hot or cold—perhaps also an hour before meals. The alkaline salts also are eliminants, particularly the potassium and lithium salts, and may be given to aid the action of the water. Certain mineral waters are useful in this respect. A more definite effect on arterial tension is obtained by mercurial aperients and iodides, presumably through their eliminant action, possibly also through their influence on metabolism. Most striking reduction of arterial tension follows the administration of a single

grain of calomel, pilula hydrargyri, or hydrargyrum cum cretâ nightly or every second or third night, with just sufficient of one or other of the vegetable or saline aperients to secure an efficient action of the bowels. Colchicum or ipecacuanha seem to enhance the vaso-dilating effect. The iodides, in addition to lowering the tension by promoting elimination, may have some resolvent effect on the chronic aortitis which impairs the elasticity of the aorta and blocks the mouths of the coronary arteries.

The mild mercurial aperients and the iodides, then, form the basis of the medicinal treatment of the cardio-vascular condition which gives rise to angina. With them will be associated tonics or other remedies which may be indicated by functional derangement of any kind. Quite the most important of these is dyspepsia with flatulent distension of the stomach. The direct vascular relaxants, amyl nitrite and nitro-glycerine, are invaluable for the relief of the paroxysms, but their influence on the blood pressure is far too brief to have any beneficial effect on the arterial tension. The nitrites and erythrol tetranitrate have a more persistent vaso-dilator effect and seem in some cases to aid materially in diminishing the frequency and severity of the paroxysms. A medical friend to whom I recommended the erythrol attributed to it immunity from attacks which he enjoyed for many years.

The next *two cases of angina without valvular lesion* are brought together as contrasts. In one a sudden fatal termination took place before any considerable change could be recognized in the aorta, illustrating early implication of the coronary arteries; in the other the aorta was enormously dilated and angina had existed for years without proving fatal.

Case 5.—A woman, aged about 60 years, whom I had seen from time to time from 1892 for gouty pain and who had well-marked Heberden's nodules, complained for the first time in July, 1896, of pain behind the sternum and down the left arm on going upstairs. She was particularly liable to this in the afternoon. She suffered from obstinate constipation and looked older than her years. The pulse was very tense, the apex-beat and right ventricle impulse were powerful, and the sounds were loud, the aortic second being accentuated. Hydrargyrum cum cretâ with extractum colchici and pilula colocynthidis cum hyoscyamo were given two or three times a week and arsenic and strychnine after food. Two months later she was much better and rarely had pain. In December, all treatment having been given up, she got up from her chair one afternoon and was walking across the room an hour after rather a large meal of minced chicken and mashed potatoes when she fell forwards dead.

CASE 6. *Angina; dilated aorta.*—A City man, aged 58 years, complained of gradually increasing pain in the cardiac region on exertion, shooting to the back and left shoulder and down the left arm. He has now to stop after walking 20 or 30 yards. The pain is more easily induced after food. He can lie down and has no pain lying or sitting. He had been compelled to give up business eighteen months previously on account of the pain, and had suffered from it for about five years altogether. Except that he was pale he did not look ill. The pulse was sudden, short, and unsustained, suggestive of aortic regurgitation; the artery was large and thin-walled, not emptying when the hand was raised. There was no visible carotid throb but the brachials were tortuous and visible at the bend of the elbow. No impulse or apex-beat could be felt. The dimensions of the heart were not much altered, but there was dullness for three quarters of an inch to the right of the upper sternum, and here the second sound was loud and ringing with a musical tone. This second sound could be followed across the manubrium to the pulmonic area, showing that the aorta was dilated and thin-walled along the whole of the arch. The first sound was short; the tracheal breath sounds were conducted to the manubrium by the enlarged vessel but no tracheal tug was detected. A month later, after taking iodide of potassium (three grains), liquor arsenicalis (five minims), with tinctura cinchonae composita and spiritus aetheris nitrosi, and resting more, he had much less pain, but on exertion he had perfectly characteristic angina. The physical signs were the same but a slight tracheal tug could be felt and the left jugular vein was full and did not empty on deep inspiration so that the innominate of this side was compressed.

The following case is given to illustrate *angina from syphilitic disease of the aorta.* It was not dilated and the blood pressure was not high.

CASE 7.—A man, aged 59 years, consulted me in June, 1899, for pain, which, according to his own account, was always latent in his left arm about the elbow and was roused by slight exertion and was then felt in the cardiac region and shot up the neck into the shoulder and down the arm. It had been very severe and brought him to a standstill when walking. The pulse was weak and unsustained though the artery was full between the beats. It varied in volume and force. The dimensions of the heart were normal; no impulse or apex-beat could be felt; the sounds were weak, the first was short, and the aortic second was scarcely audible; there was an indistinct reduplication of the second sound over the right ventricle. He was a *bon vivant*, but before I saw him had been cut off smoking, coffee, and brandy. There was a history

of syphilis. A blister had been applied and he had taken potassium iodide with strychnine and spartein. Benzo-naphthol had also been given for the relief of flatulence. The pain was less severe than it had been, but he always ceased to walk when it came on. He looked well and had a good appetite; he slept well. The iodide was continued in five-grain doses with arsenic, ammonia and bark, and pilula hydrargyri (one grain) with pilula rhei composita and hyoscyamus was given twice a week. Nitro-glycerine tabloids were taken when the pain was severe. I saw him at long intervals. No change of note took place in the symptoms or physical signs. After a time slight albuminuria, which appeared fitfully at first, became permanent, the specific gravity of the urine being about 1014. I saw him last in December, 1902, by which time he was confined to his room and he died suddenly shortly afterwards.

Fatty degeneration of the heart.—The opinion has been entertained that the condition of the heart commonly found after death from angina was fatty degeneration in association with calcareous degeneration of the coronary arteries. It is only in a certain proportion of the cases, however, that there is the advanced degeneration in which the fingers sink into the softened walls and the heart almost falls to pieces by its own weight. The physical signs of fatty heart are chiefly negative. The dimensions are normal unless fatty change has supervened in a heart previously hypertrophied or dilated. No impulse or apex-beat can be felt; the first sound is short and weak; the second, both aortic and pulmonic, is also very weak. The interval between the first and second sounds may be shortened, the ventricle not going through with its systole. The rate may be slow. All these conditions, except, perhaps, the approximation of the first and second sounds, may be present in cardiac asthenia, particularly if the heart is overlapped by the lungs. Sometimes a fairly good second sound is so masked when the lungs meet in the middle line that it is scarcely audible, but it will be heard in the carotids. We do not, therefore, make a hasty diagnosis of fatty heart. The pulse may be of great assistance. A sustained pulse at once negatives advanced fatty degeneration. On the other hand, calcareous degeneration of the radials or a thin inelastic condition of the arterial walls will be confirmatory of degeneration of the heart. Paralysis of the diaphragm will be evidence in the same direction. Fatty heart and fatty diaphragm not infrequently go together. It has seemed to me that the paroxysms last longer in fatty degeneration of the heart. The two cases mentioned of agonizing pain going on through the night were examples of this condition.

I have spoken of *dilatation of the stomach* as capable of giving

rise to attacks closely simulating the paroxysms of true angina. This condition may in cases of angina or of any serious heart disease precipitate the final event and be the immediate cause of a fatal issue. Naturally, also, it may contribute to bring on attacks, as indeed, may a hearty meal. In almost all cases angina is induced more readily by exercise soon after food. This makes it very important that the upper limit of the gastric resonance should be carefully ascertained in all cases of angina.

I have actually witnessed a death due to dilatation of the stomach. I was asked to see a patient a short distance from town. From the description of the case, which was clear and accurate in every detail, it was obviously one of true angina and probably of fatty heart. The stomach was seen to be distended, and in the course of the examination it had just been pointed out that the upper line of resonance corresponded with the fifth rib and the base of the ensiform when the patient became slightly convulsed and unconscious and died before our eyes.

Another case, a man, aged 73½ years, was seized with pain in the chest and shortness of breath on his way to the city on November 28, 1894, and turned aside to consult me. He had shot during the autumn, and the first time he had any symptoms of the kind was not more than ten days previously, but from the 25th he had had pain, shortness of breath, and a sense of hollowness on walking each day. The dimensions and sounds of the heart were normal, the stomach resonance came very high and was extensive. He went into the city against my advice and later in the day took a railway journey into the country. He died suddenly on going to bed that night after a heavy dinner.

CLINICAL SIGNIFICANCE AND THERAPEUTICAL INDICATIONS OF VARIATIONS IN THE BLOOD PRESSURE

British Medical Association, Toronto and Edinburgh (*Brit. Med. Journ.*, 1906, 2, and 1898, 2)

In order that the clinical significance of variations may be appreciated, the physiology of blood pressure must be understood.

Physiology of Blood Pressure.—The entire purpose of the circulation is primarily nutrition of the tissues and the provision of the various secretions, which are anabolic operations, together with the supply of material for the chemical katabolic processes by which heat and the muscular and nervous forms of energy are evolved. It is by the capillaries that the distribution of nutrient material takes place, and it is only when outside the capillaries that it becomes available for tissue nutrition. While gases and crystalloids can pass through the capillary walls by osmosis, the transudation of liquor sanguinis, as finally shown by the experimental investigation of Professor Starling, can only be effected by pressure. There must, therefore, be intracapillary pressure, and, in order to maintain the degree of pressure in the capillaries necessary for the transudation of colloids, there must be resistance to the flow of blood in the network itself or on its distal side. A part of such resistance will be the pressure required to maintain the current of blood in the veins towards the heart. This, however, cannot be the only or the most important source of backward pressure of blood in the capillaries. It can scarcely be present in the head and neck, where gravitation and the suction action of respiration make resistance in the venous circulation almost a minus quantity; and so important an operation as the due supply of nutrient fluid to the structures could not be allowed to depend on so variable a condition as backward pressure in the veins.

It is in the capillaries themselves, therefore, that the resistance which determines the outflow of nutrient fluid to the tissues resides. This will be due in some measure to the increased size and tortuous course of the capillaries, which will make the rate of movement slower and increase the lateral pressure on their walls, but such obstruction is altogether insufficient. The viscosity

of the blood is another explanation offered of the resistance and delay in the capillaries; but viscosity is a relative term, and the passage of a fluid through a capillary tube depends not merely on the cohesion between the molecules of the fluid itself, which constitutes viscosity, but on the cohesion between the fluid and the walls of the tube. Water rises in a capillary tube of glass, but refuses to rise if the tube is greasy. Mercury, again, only enters a glass tube under pressure. It stands at a lower level within the tube standing perpendicularly in a vessel containing mercury than outside, and is convex at the end instead of concave like water.

In this sense of cohesion between the blood and the capillary walls the viscosity of the blood is the main cause of the resistance which gives rise to the intracapillary pressure. More accurately, it is the relation between the blood and the capillaries as representing the tissues which determines the resistance, and conditions of the capillary wall as well as changes in the blood may increase or diminish it.

The Capillaries.—A factor which will have a considerable influence on the pressure required for the transudation of the liquor sanguinis is the permeability of the capillary walls. Transudation of liquor sanguinis for the nutrition of the tissues being necessary, the less permeable the capillaries the greater will be the pressure required. The permeability must vary greatly in different structures. In glands called upon to furnish an abundant secretion from time to time the capillary walls will be more pervious than in connective tissue; in the lower extremities, where the weight of the column of blood both in the arteries and the veins must add to the fluid pressure in the capillaries, the walls must be less permeable than in the head and neck. It is probable that the capillary walls become less permeable in advanced life, and that capillary sclerosis may aggravate the effects of arterio-sclerosis in the production of high blood-pressure, and may indeed be the initial degenerative change. It is, again, extremely probable that differences in the permeability of the capillary wall may be the explanation of the congenital or infantile differences of arterial tension.

The intracapillary blood pressure required for the transudation of nutrient material being the primary object of the circulation, it determines the degree of blood pressure in the arterial system. The demands of the tissues, especially of the active structures, muscles, nerves, secretory organs, vary almost from one moment to another; there must, therefore, be a reserve of propulsive energy available in excess of what is required for the ordinary or average rate of

flow, and there must be a mechanism for regulating the amount of blood allowed to reach the capillaries. The pressure maintained in the aorta and large arteries by the heart is greatly in excess of what is necessary for the capillary circulation and constitutes the reserve, while the arterioles and vaso-motor nervous system constitute the regulating mechanism cutting off or turning on the supply of blood; according as the arterioles are tightened up or relaxed the blood is excluded from the tissues so that the part is pale and cold, or admitted freely, so that the part is warm and flushed. The vessels, again, are under the control of the vaso-motor nerves.

The Arterioles.—But it is not simply the supply of blood to individual parts which has to be controlled. The blood supply to different parts has to be co-ordinated and adjusted to varying conditions—to the local demand, say, for some active secretion serving the general purposes of the body, or to the increased volume and special modifications of the blood after food. This is the office of the vaso-motor system of nerves, but the blood vessels and their nerves are the servants of the tissues, and the great variations in the supply of blood are in response to tissue demands.

The blood supply, then, being determined by the requirements of the tissues, the contraction or relaxation of the arterioles will be subsidiary to the resistance in the capillaries. If, for example, for any reason the capillaries refuse passage to the blood, the integrity of the delicate network demands that the blood should be shut off, and the arterioles contract, either directly in response to the increased resistance in front or through the intervention of the vaso-motor mechanism. On the other hand, any increase in the demand for blood on the part of the tissues must be met by relaxation of the arterioles.

Variations in the resistance to the flow of blood through the capillaries are occasioned by variations of the relations between the blood and the tissues, and such variations may arise either on the side of the blood or on the side of the tissues. Perfusion experiments have shown that a great variety of substances influence the passage of blood through the capillaries of a given structure quite independently of the vaso-motor system. A very slight chemical change in the blood is sufficient. It is an interesting fact, recently demonstrated by Professor Starling, that while the secretion of gastric juice is greatly affected by the psychical reflex of the sight or odour or taste of food, as shown by Pawlow, the pancreatic secretion is determined by a chemical reflex, the presence in the blood of a substance to which the name of secretin has been given; in other words, by a change in the relations between the

blood and the structures. The internal secretion of the thyroid gland, again, relaxes the arterioles, but the myxoedema which follows the withdrawal of this secretion shows that the primary influence of the thyroid secretion must have been nutritional, a tissue reaction to which the relaxation of the arterioles was secondary.

The blood pressure has thus to be considered in two aspects, as it is present in the capillaries, where it determines the transudation of liquor sanguinis for the nutrition of the tissues, and as it exists in the arterial system behind the arterioles. This last has been studied and discussed as if it had an intrinsic and independent importance of its own, whereas it is subservient to the intracapillary pressure, and derives its importance from the fact that it ministers to the nutritional operations.

We have thus established a basis for the clinical significance of variations in the blood pressure. It is essentially the presence in the blood of some normal or abnormal constituent.

There may, of course, be increased arterial tension from external cold, lowered tension from external heat, and there are nervous variations of an extreme character, such as the temporary tightening up of the arterioles from excitement with acceleration of the action of the heart, or the persistent small pulse of hysteria or other neuroses; or, on the other hand, the cardio-vascular atony of neurasthenia and debility. These variations, due to nervous causes, we have not time to dwell upon and must set aside, not forgetting, however, that affections of the nervous system, such as melancholia, may have a toxic origin, a clue to which may be furnished by the pulse.

The Pulse.—It is by the radial pulse that we estimate clinically the pressure in the arterial side of the circulation, and it affords to the educated finger information which intelligently interpreted goes farther and is more valuable than the instrumental registration of the blood pressure. One of the first questions to be determined is the criterion of normal blood pressure in the radial artery.

The character of the pulse indicative of normal blood pressure, or rather of a normal flow through the capillaries, can only be arrived at by observation.

As felt, the vessel, which is flattened under the fingers after the wave has passed, fills quickly, but evenly and gently, and assumes the cylindrical form, as the pressure within it is increased by the blood injected into the aorta; after a very brief period, during which the artery remains full and distended, the internal pressure gradually diminishes, and the vessel *pari passu* allows itself to be flattened, the fall of pressure being interrupted by a secondary wave started by the elastic rebound of the aorta, so slight that it

can scarcely be felt. This is represented by the familiar sphygmographic tracing, a steep but not quite perpendicular upstroke, a rounded summit, a gentle descent broken about one-third of the way down by the dicrotic rebound and notch. The normal blood pressure is not represented by a given number of millimetres of mercury ; so long as the beat of the pulse, as felt, and the sphygmographic tracing are of the character described they must be accounted normal. The artery may be small or large, and the beat may be weak or strong, according as the systole of the heart is vigorous or languid, but so long as the vessel allows itself to be easily emptied and flattened between the beats, there is no undue resistance to the flow of blood through the capillaries.

There are, of course, physiological variations on each side of the average pulse. In some types of constitution the arteries are small and the blood pressure low, so that the wave is easily arrested and the pulse extinguished, while the vessel cannot be felt between the beats. This pulse is common in fat people. In thin, wiry individuals the artery is usually large and the beat more forcible and distinct. Such differences are recognizable even in childhood, and may be very marked in adolescence. There would seem to be a congenital difference in the permeability of the capillary walls, so that greater pressure is required for the transudation of liquor sanguinis in some constitutions than in others.

In the clinical interpretation of the variations of blood pressure recognized in the pulse there are several questions to be determined.

The Heart.—One of the first is whether the rise or fall is to be attributed to the heart or to the arterioles and capillaries, to increased or diminished driving power from behind, or to increase or diminution of the resistance in front. Most commonly the central and peripheral influences are operating in the same direction, as in the familiar instance of resistance in the capillaries giving rise to contraction of the arterioles, the obstruction so induced being met by increased systolic vigour of the heart. In the debility following acute febrile affections, especially perhaps influenza, the exact contrary is seen, relaxed arterioles and capillaries with poor propulsive power on the part of the heart. Such accord, however, is not invariably present ; there may be powerful action of the heart, making for increased vascular pressure, and at the same time great relaxation of the peripheral vessels, tending to counteract it, as in early stages of pyrexia, particularly perhaps in pneumonia.

There is no risk of the action of the heart being overlooked or misinterpreted when it is aggravating blood pressure, but when a reduction of the intravascular pressure takes place, care is not always taken to assign to the heart and vessels their respective

share in the result. The significance of the lowered tension is totally different according as it is produced by diminished propulsive energy in the heart or diminished resistance in the periphery.

No inference from the pulse or from the pressure registered by the sphygmograph can be regarded as conclusive without an examination of the heart. The dimensions, the seat and character of apex-beat and right ventricle impulse, the character of the first sound, the relative intensity of the two second sounds, or, as it is usually expressed, the accentuation of the aortic or pulmonic second sound, respectively, the respective length of the intervals—all enter into the estimate to be formed of the efficiency of the left ventricle systole and of the volume of blood launched into the aorta, and a safe conclusion can usually be arrived at as to the way in which the heart is maintaining the blood pressure. Strange contradictions may, however, present themselves between the heart and the pulse. Overlapping lung in a deep-chested individual may disguise a powerful impulse and muffle loud sounds so that with a good pulse wave the heart may seem to be weak. On the other hand, the apex-beat and impulse may be powerful and the first sound loud when the pulse is extremely weak, if from any cause the left ventricle receive an insufficient supply of blood. Leaving valvular disease and adherent pericardium out of the question, this may arise from effusion into the pleural cavity or pneumothorax; from obstruction in the pulmonary circulation by extensive pneumonic consolidation, or bronchitis, or asthma, or so-called congestion; or, again, by extensive structural disease, tuberculous or malignant.

A less frequent cause, and one which commonly escapes recognition, is pressure on the right ventricle by the diaphragm. This ventricle is thin-walled and rests on the sloping surface of the diaphragm; and when this muscle is carried forcibly upwards, as it may be by general distension of the abdomen or by dilatation of the stomach, the elastic recoil from systole is interfered with and the pressure in the great veins fails to fill the ventricle, so that a very inadequate charge of blood is carried into the pulmonary artery and consequently to the left auricle and ventricle. The action of the heart may be vehement and the impulse powerful and the first sound loud, but if there is very little blood in the ventricle, the compression wave propagated along the arteries can only be feeble.

General distension of the gastro-intestinal canal so that the abdomen is prominent and tight and the diaphragm is carried upwards is not uncommon in acute febrile disease of various kinds, and may sensibly aggravate the danger of the attack. Dilatation

of the stomach is less conspicuous and gives rise to less urgent symptoms, but the continuous embarrassment of the heart is a source of serious disturbance of the circulation, such as breathlessness and palpitation, and may provoke attacks closely resembling angina.

In tachycardia and palpitation the small, scarcely perceptible pulse sometimes found, and the sense of faintness, may be due not to weakness of the systole, but to the fact that the diastole is so short that the ventricles have not time to fill.

The Peripheral Resistance.—The actual degree of pressure in the circulation is of course ultimately dependent on the heart, but its maintenance depends on the obstruction offered to the blood in the arterioles and capillaries. If they are relaxed so that the blood shoots through them without resistance as through the open end of a pipe, the tension, however violent, is only momentary, and after the beat the artery allows itself to be at once flattened by the finger. If, on the other hand, the resistance in the periphery is considerable, the artery is full between the beats and can be rolled under the finger however weak the compression wave transmitted from the heart.

This peripheral resistance, therefore, is a most important subject of clinical study, and its estimation and interpretation constitute the significance of the clinical variations of the blood pressure. The physiology of the blood pressure has already been discussed, and, while it is evident that the degree of tension in the arterial system is immediately determined by the resistance in the arterioles, that is subservient to and governed by the resistance in the capillaries. The arterioles relax or contract in obedience to the demands of the tissues. Ultimately, therefore, the significance of variations in the blood pressure is based on the relation between the blood and the tissues, and, while high arterial tension is studied with respect to its effects on the arteries and on the heart, it should be studied still more closely as throwing light on states of the blood and changes in the structures.

Disturbance of the relations between the blood and tissues originating in the tissues is exemplified by inflammation resulting from injury. Inflammation was formerly regarded as a vascular process, and as consisting in, first, contraction, then dilatation of the vessels with initial acceleration of the blood through the part followed by stasis; but these circulatory phenomena are secondary to an antecedent change in the biochemistry of the damaged structures.

A change takes place in the capillary walls which leads to increased cohesion between them and the leucocytes, so that these

cling to the capillary, and first move slowly, then come to a standstill, and finally penetrate the wall and accumulate outside. There is no rupture of the capillary, and no aperture is left. The leucocytes pass through the capillary wall as a soap bubble can be passed through a soap film without rupture of either, and for the same reason—namely, that the capillary wall has become assimilated to the corpuscles in consistence and constitution. There is no extravasation of the red corpuscles.

Low Blood Pressure.—The most common and familiar cause of lowered vascular pressure from blood conditions is pyrexia. The heightened temperature contributes to the relaxation of the arterioles and capillaries, and the fall in pressure, but is not the determining influence. In scarlet fever with a high temperature the radial pulse may be small and tense; in inflammation of serous membranes the arterioles are usually contracted. I have seen a small tense pulse in septic pneumonia with a temperature of 104° F., pneumonia being a disease usually attended with extreme relaxation of the arterioles and capillaries.

In pyrexia, at any rate in its early stages, the increased frequency and force of the heart's action would make for increase in the blood pressure, but this is neutralized by the diminished resistance in the periphery. Obviously the whole process is a response to some increased tissue demand, local or general—local when microbic invasion or inflammation of some organ or structure requires an abundant supply of leucocytes, general when the presence of septic micro-organisms in the blood calls for the production of antitoxins by reaction between the blood and tissues.

Other causes of lowered blood pressure, when this is not due to heart failure, are various forms of debility or toxaemia. There seems in some cases to be an entire absence of the resistance in the capillary network which is necessary for the transudation of nutrient material.

High Blood Pressure.—The clinical significance of high blood pressure is generally admitted to be the presence in the blood of some constituent—a glandular secretion, or product of metabolism or mineral matter—which provokes resistance in the peripheral circulation. Such resistance is very commonly attributed solely to the contraction of the arterioles, but this would be meaningless without reference to the blood and tissue reactions which take place beyond the arterioles. It is in the capillaries that the resistance is initiated, and the arterioles and, in case of need, the entire vaso-motor apparatus, nervous and muscular, are called upon to co-operate.

High blood pressure runs in families, and we find high tension

pulse in children, and see the tortuous arteries which we usually associate with age coursing across the temples of quite young men. The states of system with which it is associated—renal disease, gout, lead poisoning, lithaemia, constipation—are all, or nearly all, characterized by imperfect elimination or imperfect metabolism of nitrogenized matter. This for the most part is the causation and the significance of the high tension pulse, the resistance being primarily in the capillary network, and the stop-cock action of the arterioles secondary and defensive. The effects are numerous and varied. First hypertrophy, and then degeneration of the walls of the arterioles and arteries, abolishing their function of regulating the local blood supply and impairing the adjustment of the circulation to varying external conditions; dilatation of the aorta and its main branches, with atheroma of the arterial tunics and loss of the elasticity which converts the intermittent propulsion of blood by the heart into a continuous current, thus approximating in character the radial to the aortic pulse; hypertrophy or dilatation of the heart, or both, or fibroid or fatty degeneration, with their respective consequences; thickening and deformation of the valves with obstruction or incompetence. Other effects, structural or functional, too numerous to specify, and too familiar to need description, flow from these changes in the heart and vessels.

While it would be waste of time to go over the old, well-trodden ground, the variations in vascular pressure are of almost universal clinical application, and to some of these I may be permitted to allude.

Indications for Treatment.—For many years I have been accustomed to take as one of the chief guides in the prognosis and treatment of the multitudinous functional derangements of the nervous system which have come to be grouped under the name neurasthenia, the plus or minus tension presented by the pulse. There is more scope for treatment and better promise of relief in cases in which the vascular pressure is high than in those in which it is unduly low. When the tension is high there is the possibility that the impurity in the blood, which gives rise to resistance in the capillary circulation, may also act as a poison to the nervous system, causing a sense of intense depression and obstructing the development of nervous energy, and that this may be eliminated: a simple illustration is the effect of a blue pill, which sometimes seems to change the entire aspect of the world to the sufferer from what is called biliousness. This may be looked upon as an acute form of a condition which, when chronic, may give rise to melancholia. A most interesting paper appeared in the *Lancet* of June 25, 1898, in

which Dr. Craig, of Bethlem Hospital, has shown that in melancholics the vascular pressure is high; in maniacal excitement low, and that modification of the tension is attended with more or less of a corresponding effect on the symptoms. A valuable point in this communication is the explanation of the disappointing results of hypodermic injections of morphine in states of excitement. Instead of sleep there is often an aggravation of the excitement, which is a consequence of the lowered vascular pressure produced by the morphine. This effect of morphine on vascular tension is an important factor in its favourable influence in angina pectoris and in the relief of distress in some cases of aortic incompetence.

Among the diseases in which it has seemed to me that vascular pressure or arterial tension may be a means of discrimination between cases which require different treatment is epilepsy. The two extremes of tension are met with in association with periodical attacks of general convulsions of the epileptic type; when it is low the prognosis, according to my experience, is much more unfavourable than when it is high. The epileptic tendency is probably inherent in the nerve centres, and the fits not dependent on external influences.

Uraemic Convulsions.—I may perhaps here again express my opinion that uraemic convulsions are directly related with high vascular pressure, and are produced by arrest or disturbance of the capillary cerebral circulation, and not by the mere presence in the blood of any urinary poison. Uraemic convulsions do not follow ligature of the ureters, and cannot be produced experimentally by injecting urea or any of the renal derivatives. They supervene at periods of renal disease, when the accumulation of such poison cannot be great, and often fail to come on when the uraemic saturation has obviously reached an extreme degree. Convulsions of identical character are sometimes seen when there is no renal disease at all, but high arterial tension due to some other cause. A medical man is still alive and well whom I bled to 30 ounces, 20 years ago, for severe convulsions; he had extremely high arterial tension, but never at any time presented indications of disease of the kidneys, and he had never had syphilis.

Chronic Bronchial Catarrh.—Another affection in which the clue to treatment is often furnished by the degree of vascular pressure is chronic bronchial catarrh, especially, perhaps, when it is associated with asthma. We are all familiar with the term gouty bronchitis, and this need not be a mere refuge for ignorance, or a convenient phrase for the satisfaction of the patient and his friends in the presence of intractable disease. In many of these cases of recurrent bronchial catarrh the pulse is one of high tension, and

the blood is charged, if not with uric acid, with some nitrogenized waste which in some way or other predisposes to catarrh of the bronchial tubes. In such cases, while attacks may be warded off by wintering in the south, much more effectual relief may be afforded by eliminants.

Cheyne-Stokes Respiration.—I should like also to point out once more the association of Cheyne-Stokes breathing with high arterial tension. In the course of many years' observation, extending now over hundreds of cases, I have never seen true Cheyne-Stokes respiration without high intravascular pressure. But first let us understand what we mean by the term, and I may remark in passing that it is not the deadly symptom it is sometimes supposed to be. It may add to the prognostic significance of other unfavourable symptoms, but patients in whom it has been conspicuously present for a considerable time have often completely recovered.

(1) It is not simply a suspension of respiration. In meningitis and other affections of the brain the breathing stops slowly or suddenly, and is suspended for a longer or shorter time, beginning again suddenly and going on rapidly, but there is no regularity about it. If we listen to the breathing of an aged person it will often become slower and more embarrassed till it stops, sometimes for what seems an alarming time, and then starts with a loud snort and deep sigh. Neither of these is Cheyne-Stokes breathing. In Cheyne-Stokes respiration there is a cyclical recurrence of a complete pause which may last ten, fifteen, or twenty seconds, or even more, after which the breathing returns, at first scarcely perceptible, but gradually increasing in depth and vigour till the movements greatly surpass those of normal respiration, and they die down just as gradually.

(2) There is no cyanosis during the pause, or very rarely anything which can be called dyspnoea, and the patient very rarely notices or complains of the breathing. In only one case have I seen serious respiratory distress; in another I thought the patient, an old medical man, talked himself out of breath when he simply took advantage of the pauses to speak, which he was unable to do when breathing.

A very instructive case came under my observation many years ago. I was called to a patient who had had a severe cerebral haemorrhage with left hemiplegia. He was lying on his back breathing stertorously but quite regularly, and was deeply cyanosed from the stertor. I turned him upon his side, as we were taught to do by Dr. Bowles, the stertor ceased, the cyanosis rapidly cleared up and then Cheyne-Stokes breathing set in.

(3) It cannot be put down to any special condition of the nervous

system. Cheyne-Stokes breathing may be present when there are no symptoms of nervous disturbance of any kind. The one fact which affords evidence of the influence of the cerebral hemispheres is that after a cerebral haemorrhage giving rise to hemiplegia there may be Cheyne-Stokes breathing which had not been induced by the intra-arterial pressure which had ruptured the vessel.

If the subject is at the point of death from renal disease—and it is in renal disease that Cheyne-Stokes respiration is most common—he may be confused or unconscious, but this is not a necessary condition; the cyclical rise and fall and suspension of respiration will have set in long before this and will continue if the cerebral symptoms pass off. I have known a patient go to the city regularly for three months and transact business who had always had Cheyne-Stokes breathing when I saw him, which was pretty often.

I have many times been told by nurses that a patient who had Cheyne-Stokes respiration when awake, breathed regularly during sleep, but I have never had the opportunity of verifying this observation. It is certainly not constant, but if it is common, seeing that the activity of the higher nerve centres is suspended during sleep, it would seem that the influence of the cortex tended to induce the cyclical breathing.

Whenever the arterial tension is reduced—unless this is simply due to diminished vis a tergo—whenever, that is, the resistance in the arterio-capillary network is diminished, by whatever means, Cheyne-Stokes respiration disappears, for example in febrile conditions, when there is relaxation of the arterioles even if the pyrexia has been due to a pulmonary infarct; when the bowels have been freely opened by a mercurial aperient, if constipation has caused or aggravated high pulse-tension; when the arterioles and capillaries have been relaxed by erythrol tetranitrate or by subcutaneous pilocarpine or morphine. I have only once seen Cheyne-Stokes breathing in enteric fever, and in this case there was intense headache with early cerebral symptoms and a small, very high tension pulse.

If the logical definition of causation is invariably antecedent, high arterial tension must be set down as the cause of Cheyne-Stokes breathing.

Treatment of High Pressure.—Whether the deviation from the normal blood pressure be in a plus or minus direction the first consideration will be how far it is due to the heart. The pulse will usually afford very definite information on this point, but its indications must always be checked by a careful examination of the heart. Want of vigour and efficiency in the action of the

heart, whether associated with diminished or increased peripheral resistance, will be met as far as possible by such tonics as are indicated by the condition of the heart itself or by the general constitutional state. When, however, there is resistance in the capillaries and arterioles, a weakened heart will profit more by the reduction of the work thereby imposed upon it than by attempts to improve its tone and strength.

It is rarely desirable to endeavour to reduce directly any apparent excess of cardiac action; such increase of propulsive power as may be recognizable by a powerful impulse and accentuated sounds will have been evolved to meet obstruction in the peripheral circulation, and, while it may become a source of danger, it is essentially compensatory in character.

The blood pressure, however, is almost entirely conditioned by the resistance in the arterioles and capillaries, and this, again, by the relation between the blood and the tissues. It is, therefore, to the peripheral circulation that attention must be directed in dealing with unduly high arterial tension. It is not simply the pressure as such that we seek to reduce; our endeavour should be to remove the conditions which give rise to it.

When arteriole contraction is regarded as the primary cause of the obstruction to the outflow of the blood, it is natural to have recourse to the vascular relaxants, such as nitro-glycerine, erythrol tetranitrate, the nitrites; but, while temporary relief of great value may be given, and attacks of angina may be warded off, the effect is only fugitive. The remedy has not gone to the root of the evil. Conditions of blood being recognized as underlying and determining the resistance in the capillaries and arterioles, it is to them that therapeutic measures will be addressed.

Some of the substances present in the blood which give rise to resistance in the capillaries, such as the secretions of the suprarenal bodies and of the kidney cortex and of other glands, are not under our control. On the other hand we can easily withdraw calcium salts, sodium chloride, and other saline matters which directly or indirectly appear to have this effect, and can avoid giving certain vegetables. The principal problem which faces us is how we can influence the metabolism by which food, after absorption into the blood, and after forming part of the various structures in the course of the breaking down of the large molecules, and of the hydration and oxidation by means of which energy is evolved, may be brought into chemical combinations suitable for elimination.

Here, of course, diet comes in. If we give food which either from quantity or quality is incapable of easy and complete assimila-

tion and oxidation obstruction in the capillary network takes place. It is not necessary to repeat the admonitions to simplicity and moderation which one reads everywhere; the food must be adjusted to individual requirements. I would only say that I do not share the horror of purin-containing articles of diet which is now the fashion. Most of us digest and assimilate them without difficulty. An aid to elimination to which I attach great importance is the drinking of a tumbler of water not impregnated with lime salts night and morning.

Turkish baths, by inducing copious perspiration are powerfully eliminant, and the massage and frictions which form part of the procedure aid in this. Warm baths and other baths are also useful. Exercise in the open air, carried to the point of free perspiration, not only brings about elimination, but has a still more important effect in promoting metabolism and oxidation.

With regard to medicinal therapeutics, again, little need be said. The eliminant action of the numerous mineral-water resorts to which lithaemic subjects are sent is well authenticated, and we take daily advantage of alkalies and of saline and other aperients. The primary effect of alkaline waters and of the alkalies and salts administered as medicines is mainly if not solely solvent and eliminant. Their influence on metabolism is secondary through the removal of waste products which interfere with it. If we take uric acid as representing the materies morborum, alkalies dissolve and copious water carries it off; they do not prevent its formation. In my judgment the various preparations of mercury go further than this and have an influence on metabolism. I can, at any rate, state from observation extending over forty years that mercurial aperients have a definite and constant effect on the blood pressure. Not by reducing the driving power of the heart, but by diminishing the resistance in the peripheral circulation, and the effect on the pulse is not at all in proportion to the aperient result, but may be manifest when the action of the bowels is lessened rather than increased.

Treatment of Low Pressure.—In the general management of unduly low vascular pressure the first object will be the identification of the underlying vice of nutrition. This may be some derangement of the digestion or of the process of assimilation, or there may be failure or perversion of one of the glandular secretions which have been found to play so important a part in nutrition, such as the suprarenal or thyroid, or organic disease may be destroying some important organ or undermining the vital energies. Cancer, for example, proves fatal not merely by its local effects, but by pouring into the blood some deleterious material; or a primary

affection of the nervous system may exercise an unfavourable influence on nutrition and on the circulation. The cause having been ascertained, the chief treatment will be such as is directed to its removal. The cardio-vascular tonics, digitalis and the like, are often useful accessories, although they deal only with effects and not with the cause of the low tension.

EXTRACT FROM A CLINICAL LECTURE ON ANEURISM OF THE AORTA

Delivered at St. Mary's Hospital

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THE diagnosis of this disease varies very much in difficulty; sometimes it is obvious, at others most difficult; it may be easy or the reverse, in the same case, at different stages, as in Andrews, where the signs were extremely obscure at first, whereas at present the diagnosis may be made with the greatest ease. I have now a man under my care who has up till quite lately been doing extremely hard work, and who, although I believe him to be suffering from aneurism, was sent into the hospital for other reasons; the diagnosis of aneurism in this case is chiefly based upon a remarkably loud and voluminous second sound and inequality of the pulses. In many cases the aneurism is never discovered, and perhaps is not discoverable: for instance, there may be a small aneurism at the root of the aorta bursting into the pericardium, or an aneurism of the transverse arch pressing backwards on the trachea, producing ulceration of the cartilages and bursting into the trachea, or an aneurism of the descending aorta bursting into the pleural cavity. I have made post-mortem examinations in cases of sudden death from each of these causes, the patients having been apparently in good health up to the moment of the rupture. If the aneurism projects from the anterior wall of the descending aorta, the diagnosis is often very difficult; if from the posterior wall, it is usually more easy.

Physical Signs.—There is nothing absolute in the symptoms or signs of aneurism. In considering the diagnosis, symptoms must be put together and an opinion must be formed from them collectively. You may or may not have *dullness*, but an aneurism may be of some size and yet no marked dullness may be found, from its being surrounded by lung; *pulsation*, if present, is nearly conclusive, but it is often very obscure, and may require artifices to show it, such as planting the small end of your stethoscope firmly upon the chest, when, if pulsation be present, the movement will be magnified at the free end. Often when no pulsation can be detected,

a sensation of "shock" is imparted to the hand. In some cases pulsation will be obvious during expiration, though it cannot be detected during inspiration, the aneurism becoming uncovered by the retraction of the lung during the former movement. I remember well one case of aneurism projecting from the right side of the ascending aorta in which pulsation during expiration was the only positive sign of its existence. When seeking pulsation, the finger should always be pressed deeply into the supra-sternal notch; here you will sometimes find a pulsating tumour which you could not reach in any other way. This observation, however, is liable to fallacy. The arch of the aorta may come within reach of the finger here, especially when elongated and dilated in advanced life, or from aortic regurgitation, and I once met with a case of abnormal origin of the right subclavian, where this vessel crossed over the root of the neck, from left to right, and could be felt in this situation. With pulsation you may have *vibration* or *thrill* imparted to the hand.

Again, there may or may not be a *murmur*. In the case of Andrews, though a faint systolic murmur was heard at first when he stood up, this ceased to be audible for a long time, but there is now one behind. Murmurs are never to be relied upon; they are easily produced by pressure, and are often present in the subclavian arteries, but a murmur heard in the left carotid and subclavian and not in the right may be corroborative of other signs of aneurism between the innominate and left carotid. A much more valuable sign of aneurism is the *modification of the aortic second sound*, to which I have referred. This phenomenon is very difficult to describe; the sound is more drum-like and voluminous than normal, and is heard over a greater area than is usual with the second sound. When well marked, it is accompanied by, or is identical with, the "diastolic shock." This I regard as one of the most important signs of aneurism; it was first brought prominently forward by Dr. Sibson, who showed that the second sound of the heart was produced, not by the valves only, but by the sudden tension of the aortic walls. There is no such thing as a "click" of the valves. When, therefore, the aorta is dilated or has an aneurismal sac in connexion with it, the second sound is intensified and lowered in pitch by the greater extent of the resonant membrane.

The production of sound in this manner may be well illustrated by suddenly tightening a sheet of paper or piece of linen; the larger the area of paper or linen suddenly made tense, the louder the sound produced, the lower the pitch, and the greater its intensity. I know of no single sign which is of so great value as this altered and

intensified second sound. Of course in aortic regurgitation this symptom is lost, for the reflux of blood which is permitted prevents the aortic walls from becoming tense.

Another important and valuable sign is the *modification of the pulse*. This may be general or local. Wherever the sac occurs, even if it be in the abdominal aorta, an effect will be produced in the pulse in the radial artery. This general effect consists in a diminution of the percussion element of the pulse wave, the aneurism absorbing the force of this wave. But if the innominate or subclavian come off from the sac, a great change may be produced in the corresponding radial pulse, which may become extremely feeble, and, at the same time, delayed, or may, perhaps, disappear, altogether, as in two cases previously narrated, the artery remaining full and the circulation being fairly maintained. In one case, that of Bridgland, the post-mortem examination revealed a valve-like opening of the subclavian into the aneurismal sac, in the wall of which the artery ran for more than an inch; here the pulsation was entirely neutralized, for when the sac was distended the valvular opening was closed, as the sac contracted the valve opened and blood passed down the vessel. The value of this symptom is very great. I should have thought it pathognomonic had I not lately had a case under my care in the wards in which no pulsation could be detected in either radial, although the vessels remained full of blood. This proved at the post-mortem examination to be due to certain abnormalities of the vessels, causing narrowing of the commencement of the innominate and left subclavian; this had given rise to increased strain at these points, and atheroma had been produced, still further obstructing the passage of blood through the vessels and producing the anomalous symptoms referred to.

Symptoms.—The indications which we have considered thus far are those which are obtained from the circulatory system only, the remaining symptoms may all be classed as pressure effects. These are very variable in their character; they are most marked in sacculated aneurisms, and are very slight in general dilatation of the aorta.

First among them may be mentioned *pain*, which is very variable in its intensity, both in different cases and in the same case at different times; it is most severe when a nerve is caught between the aneurism and bone. This occurs most frequently in aneurisms in the descending part of the arch.

Secondly, we may have *obstruction in veins*, especially in the vena cava or the innominate veins. I have met with one case in which sudden complete obstruction of the superior vena cava was

the first symptom which indicated an aneurism, and caused the patient to seek medical advice. In this instance there was enormous distension of the head, neck, and upper extremities, the face especially being swollen and purple.

Thirdly, we may have signs of *pressure on the root of the lung:* if the aneurism be one of the ascending aorta, the root of the right lung will be involved; if of the descending aorta, that of the left. The signs of this complication will be as follows: there will be at first good resonance on percussion, but silence on auscultation, no respiratory murmur being audible; gradually dullness, due to congestion, will supervene; this will be attended by spasmodic cough and fibrinous expectoration, tinged with blood; the cough will often be very violent in such cases.

Fourthly, there may be signs of *pressure on the trachea*, producing tracheal breathing; this will especially accompany aneurisms of the transverse part of the arch.

Fifthly, *the oesophagus* may be subject to pressure, either direct or indirect, producing difficulty of deglutition.

Sixthly, *pressure on nerves* may produce symptoms which will vary according to the nerve pressed upon.

I have never seen the phrenic involved, although it would appear likely to become so; on the other hand, the pneumogastric is very frequently affected. This is indicated by nausea and vomiting, cough, spasm of the larynx, and sometimes it leads to pneumonia. If the aneurism be one of the transverse part of the arch the left, if of the right subclavian the right recurrent laryngeal may be involved, producing paralysis of the corresponding vocal cord and imparting a cracked character to the voice and giving rise to dyspnoea, paroxysmal or habitual. Dr. George Johnson has called attention to the interesting fact that pressure on one recurrent laryngeal nerve and pneumogastric may be attended with bilateral paralysis of the laryngeal muscles, which he explains by reference to the bilateral association of the vagus nuclei in the medulla.

Again, the sympathetic may be pressed upon and contraction of the corresponding pupil will then be produced. Lastly, one or more intercostal nerves may be compressed against the ribs, producing well-localized pain, and, if they become divided, loss of sensation in the part supplied by the nerves affected.

The signs and symptoms enumerated will be grouped differently, according to the part of the arch affected.

At the *root of the aorta* aneurism rarely attains large size and is made out with great difficulty, and rupture often occurs before its existence is discovered. The prominent symptoms are usually

those of disturbance of the heart, with more or less displacement and modification of the sounds.

Aneurism of the *ascending aorta* usually projects to the right. From the superficial position of this part of the vessel, dullness, pulsation, diastolic shock, and aneurismal modifications of the sounds can generally be distinguished without much difficulty. The right pneumogastric and the vena cava superior, with its innominate tributaries, are liable to pressure. The circulation in the innominate artery may also be interfered with, and with it the radial or carotid pulse.

When the aneurism springs from the *transverse part of the arch* where the aorta recedes from the surface, the signs usually become less prominent, though an aneurism of this part may reach the front of the chest and present behind the sternum or below the left clavicle. But the symptoms are often more varied and severe from the important structures liable to be affected by pressure: the trachea and bronchi, especially the right, and the root of the right lung; indirectly, the oesophagus, the pneumogastric, and recurrent laryngeal nerves. Any of the great vessels arising from the arch may be implicated.

Aneurism of the *descending arch* usually, though not invariably, tends backwards, and, being surrounded by lung, does not reveal itself by conspicuous signs. Pain from pressure on the intercostal nerves, cough, dyspnoea and alteration of the voice from pressure on the left pneumogastric and recurrent laryngeal nerves, displacement of the left lung and evidence of pressure on its root, with, in many cases, obstruction of the subclavian or carotid artery, and occasionally of the left innominate vein, are among the characteristic symptoms.

This concludes all I have to say to you upon the course and symptoms of this disease, but, before closing the lecture, I will add a few remarks upon its cause and the method of treatment to be adopted. I shall not enter fully into the *causes of aneurism*, but I may briefly state that the disease may be due either to disease of the vessels or to strain, and frequently to both. The diseases of the vessels which most frequently give rise to it are syphilitic arteritis and atheroma. It has been pointed out by Mr. Myers and others that soldiers are especially liable to this disease, owing to the strain upon the large vessels of the thorax produced by carrying their accoutrements, particularly the cross-belt, but it has also been suggested that the frequency of aneurism among soldiers may be due to the frequency of syphilis. Others who are exposed to frequent violent straining efforts, which are usually preceded by a deep inspiration, and holding the breath, such as lifting heavy weights,

etc., have a similar liability to the disease. Violent athletics, especially prolonged efforts in running or rowing, have also been known to produce it.

Treatment.—In order to carry out a rational treatment of any disease, it is necessary that we should have a clear understanding of the object to be attained, and of the means by which we endeavour to attain it. The main object in aneurisms, which cannot be dealt with surgically, must obviously be to keep down arterial distension; anything which tends to increased tension or distension of the arterial system must necessarily tend to increase the size of the aneurism, in fact, to produce the reverse effect to that which we desire—namely, to allow the aneurism to contract and a clot to be formed in the interior of the sac. Your first object, then, will be to keep the heart quiet, and for this purpose the patient must be confined to bed, and not allowed to make the least exertion. He must be carefully guarded against changes of temperature. Cold applied to the skin produces contraction of the superficial vessels, and, therefore, increases the amount of blood contained in the large vessels within the thorax; the application of cold to even a small space of skin is sufficient to produce increased arterial tension. The ingestion of a large quantity of food, again, at one meal, produces a very considerable increase in the amount of blood contained in the vessels; the patients, therefore, should take food in small quantities at frequent intervals, rather than meals at longer intervals and consisting of larger quantities. The food should also be taken as dry as possible, and the quantity of fluids should be strictly limited. Above all things, the bowels must be kept freely open, for any straining efforts are extremely dangerous and injurious, and, in addition to this, constipation increases arterial tension. If the pain be severe it must be relieved by opium. Where our hope of cure is so small, we have no excuse if we allow the patient to suffer; subcutaneous injections of morphia are especially useful for this purpose, and, judiciously employed, undoubtedly assist, directly and indirectly, to prolong life. Sometimes, in severe cases, bleeding will afford relief, but it is only temporary.

Various methods for inducing coagulation of blood in the sac have been devised; among them may be mentioned galvano-puncture of the sac by fine needles, with a view to produce coagulation of the blood around them. This was first performed in Italy, and the operation has in a few cases given satisfactory results. It is not yet a settled question whether the needles introduced should be connected with the negative or positive pole. Again, the introduction of a large quantity of iron wire into the sac has been tried with a similar view, but with no great success. The distal vessels

of the subclavian and carotid have been tied; this operation has been attended with success in some cases, and we hoped to have seen a similar result produced by natural means in the case of Andrews. But these surgical methods of treatment, besides being uncertain in their results, are applicable in few cases, and we have to consider what can be done by drugs.

One agent which has been employed is ergot; it was adopted in some of the cases previously related. The effects of ergot were much studied by Dr. Sibson. The physiological effect of ergot is to cause contraction of the muscular coat of the arteries. Dr. Sibson, so far as I understood his rationale of the treatment, assumed the presence of this structural element in the walls of the sac, and tried to induce contraction of the sac by the action of this drug. But from the fact that ergot increases arterial tension, this treatment has been described as trying "to burst the aneurism." As a matter of fact, the immediate result produced is beyond dispute; a large dose of the drug invariably reduces the size of the tumour and the pulsation in it. I have seen it under many conditions produce not only diminution in the pulsation, which might be simply a result of general arterial pressure and of sustained tension in the sac, but also in the size of the tumour. It is difficult to see how this result is brought about, for it can scarcely be supposed that muscular fibres are present in the wall of the sac. The only explanation I can see is, either that the action of the heart is enfeebled by diminution of the supply of blood to its walls, in consequence of the contraction of the coronary arteries, under the influence of the ergot, or else, that the walls of the heart itself are contracted and the cavities made smaller, so that less blood is thrown into the aorta at each pulsation. However striking the immediate results of a large dose may be, I have never seen it cure the disease.

But there is one treatment which I now invariably adopt, and I have twice seen it produce a cure; it consists in the administration of large doses of iodide of potassium. It was introduced by Dr. Balfour, and has been chiefly used in Edinburgh and Glasgow. This drug does not apparently effect a cure in the ordinary manner, by causing laminated coagulation of the blood, but by some action on the walls of the sac, which causes it to shrivel and contract. I will relate to you one case in illustration of this:—A patient, named Lucas, came under my care in the hospital during last year; she was a woman 40 years of age, of irregular habits, and engaged in hard work. When admitted into the hospital she was suffering from shortness of breath, and was unable to lie on the left side. The pulsation of the upper part of her chest was so great as to be visible through her clothes. When the chest was exposed the pulsa-

tion was seen to be present below the left clavicle and over the upper part of the sternum. This part of the chest was, in fact, pushed forward by a pulsating tumour, in which was heard a loud systolic murmur. There were signs of compression of the left lung, namely, complete silence with normal resonance; there was compensatory respiration on the right side. During my holiday last year she was treated by ergot, but no effect was produced. On my return to work, I began immediately to give thirty grains of iodide of potassium three times a day. Up to the beginning of October there was no improvement; but, on the contrary, further signs of compression of the left lung, dullness on percussion, tubular breathing, incessant and violent cough, with expectoration of much blood-stained glutinous fluid. She now began, however, gradually to improve. Little by little the prominence subsided, the pulsation became circumscribed and less distinct. On November 14, the notes state that, instead of a tumour below the left clavicle and the sternum, there was only shock, and no heave could be detected; the systolic murmur was still present. There was fair resonance, and the breath sounds were audible over the left lung to some extent; but the breathing was still tubular over the scapula. The aneurism was now practically cured, and she was allowed to get up. When she found herself well she became restive and troublesome, wandered about the hospital heedless of restraint, and one day when called to order by the sister, she was abusive and violent in the extreme, used threats to the resident medical officer and other officials, so that she had to be dismissed for disorderly conduct. She resumed her old life, and six months later she dropped down dead, no doubt from rupture of the diseased aorta.

ON VENESECTION

A paper read before the Harveian Society, February, 1880, *with slight additions*

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The disuse of bloodletting as a remedy has so frequently been the subject of remark that observations respecting it except by way of explanation, are unnecessary.

During some years there has been a gradual reaction. It has been shown, notably by Sir James Paget, that venesection is attended with very little risk either of immediate or remote injury, while on the other hand it is in suitable conditions a remedy of striking power. A return to indiscriminate bleeding would be a great evil, but of this there is really no danger. It is now better known what abstraction of blood can do and what it cannot. The indications for resorting to it can be more precisely defined, and sooner or later the teachings of physiology applied to the rectification of abnormal conditions present in disease will remove the stumbling-block for a time thrown in the way of this powerful therapeutic agency by morbid anatomy.

In the first place, general bleeding is not employed as a remedy for inflammation as such. We have in the writings of our predecessors a vivid description of the effects of venesection in iritis—the relief of pain, the diminution of vascularity, the arrest of destructive changes; but true as this undoubtedly is, such advantages, even were they constant, which is by no means the case, may be purchased too dearly, and there is no inflammation of an internal organ in which venesection is resorted to. Nor is it for the control of pyrexia, whether the result of a local inflammation or of one of the specific fevers. What we seek to do, and what we can effect by venesection, is to modify the distribution of the blood and the pressure within the arterial or venous system. The circumstances under which this may be required are various; some of them I shall enumerate, and, when I can do so, illustrate by recent experience of my own.

Aneurism.—Perhaps the most simple and unmistakable indication for the necessity of reducing the blood-pressure within the arteries

by bleeding is when an aneurism is giving rise to severe pain, or is threatening life by pressure upon a nerve or some important organ. I have repeatedly seen immediate and striking relief afforded by a small bleeding in the practice of the late Dr. Sibson, whose teaching and example I have followed. An opportunity occurred last year, when a patient, who had been some time under my care in St. Mary's Hospital suffering from a large aneurism of the arch of the aorta, was suddenly seized with violent dyspnoea during my visit to the wards. His face was purple and swollen, and he was evidently in agony, while it seemed as if he must die within a few minutes unless relief could be given. My resident medical officer, under my supervision, bled him to about eight ounces, and he was very soon in a comfortable sleep. In aneurism, however, bloodletting can only be palliative, and though it may relieve pain and prolong life for a time, it does not restore the sufferer to health. Much more than this is claimed for venesection in another class of cases when it is practised for the relief of over-distension of the right side of the heart. The circumstances under which this condition arises are various, but they are chiefly comprised under disease of the heart, or obstruction to the pulmonary circulation by disease of the lungs, especially pneumonia and bronchitis.

Pneumonia.—Pneumonia has been the great battle-ground of many therapeutic controversies, but especially of the question of bleeding and the use of stimulants. A more unsuitable field could not have been chosen. It is a disease which varies extremely as to the danger attending it at different times and places, and under different circumstances, quite irrespective of the treatment pursued, so that statistics are fallacious. Again, it runs a definite course and terminates by a very marked crisis, which allows an enthusiast or partisan to imagine that the remedy which coincides with the crisis has brought it about. The clinical history of pneumonia, uninfluenced by any but hygienic treatment, having been carefully followed and ascertained, it became possible to estimate the effects of bleeding, and it may be stated confidently that in some cases it is most useful, preventing suffering and saving life, while in others it would only be injurious. The reason is that it is not the inflammation of the lung which is combated, but a particular complication—*over-distension of the right side of the heart.* The merit of making this clear and of enabling us to recognize the indication for bleeding in pneumonia is, I believe, due to Dr. Markham. In some cases, then, of pneumonia, the rapid consolidation of a certain part of the lung with congestion of a still larger portion gives rise to a degree of obstruction in the pulmonary

circulation which embarrasses the right ventricle; this, no doubt, being enfeebled by the high temperature of the blood, induces dilatation, and eventually almost paralysis. The symptoms attending this condition, which may be established very early in the attack, are most distressing: the patient is pale or livid, instead of flushed, is gasping for breath, and probably unable to lie down; the alae nasi are working; he can scarcely speak, and the cough, if any be present, is a mere short hack, raising no expectoration; there may be a cold sweat on the face. On examining the heart, it is found to be beating violently, and a striking contrast is presented between the force of its impulse and the small and weak beat of the pulse, explained by the inadequate amount of blood which finds its way to the left ventricle, in consequence of the obstruction in the lungs and the enfeebled state of the right ventricle. However forcible the systole may be, it can have little effect on the pulse if there is only a scanty amount of blood in the ventricle to be driven into the arteries. The right auricle can generally be made out much beyond the right border of the sternum. It is under these circumstances that those striking effects of bleeding are obtained which have been so often described and which I have myself witnessed. As the blood flows the pulse improves, becomes fuller and stronger; the breathing becomes less frequent and shallow; the oppression is relieved; and not uncommonly the patient, finding to his astonishment that he can take a deep breath and cough without pain, expresses himself as feeling quite well. The pneumonia pursues its course under more favourable conditions, and recovery is usually the result. Cases of this kind are rare in the hospital practice of London, but I have met with a few. In the country they are more common.

Bronchitis frequently gives rise to over-distension of the right side of the heart, and I have seen venesection employed for its relief, but I have never ventured to practise it myself. In chronic bronchitis and emphysema, in which distension is always more or less present, the conditions which produced it persisting would certainly cause it to recur, and even acute capillary bronchitis is usually of such long duration that a single bleeding could not be looked upon as final. There are, however, no doubt cases in which we ought to give the patient the chance which venesection seems to offer in desperate circumstances, but I have hitherto preferred having recourse to an *emetic*, which will at the same time unload the air-passages and powerfully compress the heart, forcing the blood out of the right ventricle, and so far relieving the over-distension as to enable it to complete its systole, which is the end to be obtained.

Heart Disease.—In heart disease I have seen no case of aortic valvular affection, obstructive or regurgitant, in which venesection seemed to be called for, and in mitral regurgitation the good effects of digitalis can be relied upon with so much confidence that it claims the first trial. It is in *mitral stenosis* that bleeding is most frequently demanded. When the mitral orifice is narrowed there is a chronic condition of distension of the right ventricle, and this is liable to dangerous increase under various circumstances, while digitalis is not to be implicitly trusted. The question whether bleeding should be employed or not then arises not unfrequently in mitral stenosis, and indeed the evidences of dilatation of the right ventricle, the occurrence of tricuspid regurgitation and distinct reflux of venous blood into the large vessels, are more common in this than in any other form of heart disease. But while venesection is at times as strikingly useful in mitral stenosis as in the cases of pneumonia alluded to, I have not often employed it. I may have lost opportunities through timidity or excess of caution, but I think it probable that the hypertrophy of the right ventricle, which is established during the gradual increase of pressure in the pulmonary vessels, makes the over-distension less dangerous.

In deciding whether to bleed from the arm or not when there is mitral stenosis, account must be taken not only of the existing degree of distension and embarrassment of the right ventricle, but of the way in which it has been brought about. If it is the result of exposure to cold, or imprudent exertion, or emotional excitement in a patient previously manifesting little evidence of heart disease, then bleeding may be of the greatest service. In a lady to whom I was called when apparently *in extremis*, and who had been picked up on the stairs unconscious after emotion and exertion together, the action of the heart was so irregular, and the sounds so confused, that a diagnosis could only be arrived at by a process of exclusion. After eight ounces of blood had been taken she was greatly relieved. Next day the heart was acting more regularly, and a systolic tricuspid murmur was heard; a few days later the tricuspid regurgitation had ceased, and a presystolic mitral murmur was developed. With this change in the physical signs, which I have frequently noted, there was steady improvement, and the patient recovered, though she never regained her previous condition.

When, on the other hand, the distended state of the right heart under consideration comes on in mitral stenosis under the influence of the work and exposure of the daily life of the classes who come under our care in hospitals, the rest, warmth, food, and care which they receive constitute such a difference in their favour that bleed-

ing from the arm is rarely necessary; but I have often found six or eight leeches applied over the liver, enlarged and painful through venous congestion, of very great use. When, however, the venous stasis and failure of the right ventricle are the results of the valvular obstruction gradually developed in spite of favourable conditions of all kinds, bleeding, whether local or general, can be of little service.

The last occasion on which I have employed general bleeding for the relief of over-distension of the right ventricle was in October, 1882. A man, aged about fifty, was admitted into St. Mary's Hospital with a vague history of some acute pulmonary attack. He was suffering from obvious painful respiratory distress, looked pale and haggard, and complained of severe pain in the loins. His temperature was high, the pulse frequent and large but short, and the skin perspiring. There were some physical signs of bronchitis, but not sufficient to account for his dyspnoea. The heart was beating violently, the right auricle was distended, as was shown by dullness to the right of the lower sternum, and a systolic murmur was heard at the apex, but there was no evidence of mitral stenosis. His distress was so great and so clearly attributable to over-distension of the right side of the heart that it was determined to relieve this condition, although the cause which had given rise to it could not be satisfactorily made out. He was accordingly bled to eight ounces by Mr. Spicer, my resident medical officer. This gave some relief, but it was not satisfactory, and was only temporary. After death, which took place two days later, it was found that a large thrombus had formed in the pulmonary veins, and having become detached had got entangled in the mitral valve; fragments had also been carried away in the circulation, and had lodged in various organs. This sufficiently explained the cardiac embarrassment and the failure of venesection to relieve it.

High Blood Pressure.—In another and very important class of cases the indication for bleeding is high arterial tension. It is not, of course, desirable to open a vein whenever the blood-pressure within the arteries is greater than normal, but only when this has reached a point at which it is attended with urgent danger. If no serious or threatening symptoms are present and time is allowed, the tension can be reduced by other means. The effects of unduly high arterial tension are characteristic, and the condition is as easily recognizable as an indication for venesection as is over-distension of the right side of the heart; the explanation, however, of the symptoms by the assumed cause, and of the observed results of the treatment is not very easy.

Uraemia.—The most striking illustration of the good effects of bleeding is met with in uraemic convulsions. No one who has tried venesection in these cases can, I think, fail to recognize its immediate efficacy and its favourable after-consequences. My own experience, at any rate, has convinced me of the value of such a mode of treatment. I may, by way of illustration, mention two cases which came under my care in St. Mary's Hospital within a week of each other, early in 1879. One was that of a boy of twelve, which was exceedingly interesting for several reasons. He was admitted for violent uraemic convulsions coming on in the course of scarlatinal albuminuria. One interesting point was that the convulsions were unilateral, illustrating the fact that one-sided convulsions may result from a condition affecting both sides of the brain. He was bled from the arm to eight ounces. The convulsions became less severe and soon ceased, but were succeeded by extraordinary maniacal excitement, not mere delirium; illustrating another fact with which I had previously become acquainted —viz., that a morbid condition which gives rise to convulsion may in a less degree give rise to maniacal excitement. This passed off in about twenty-four hours, and finally the albuminuria disappeared in less than a week. The other case was that of a little girl, also admitted for scarlatinal albuminuria and uraemic convulsions, who was bled, and, as the boy, not only were the convulsions arrested, but within a week the albumen disappeared from the urine. She was readmitted on account of a relapse soon after her discharge, but again recovered.

It does not, however, necessarily follow that the *modus operandi* in the arrest of uraemic convulsions is by relief of arterial tension. This is, however, probable, as will be seen from the following considerations. The conditions present in uraemia, in addition to the high pressure within the arteries, which may possibly excite convulsions, are the contamination of the blood by urea or other urinary matters, a watery state of the blood, cerebral anaemia, and serous effusion. Now, abstraction of blood containing only its own proportion of urinous impurities cannot render that left behind more pure; it will indeed probably for the time be more impure, and will certainly be more watery from the absorption of fluid from the tissues to replace the blood lost. It can scarcely, therefore, be by any improvement in the quality of the blood, whether by removal of poisonous matters or by correcting its dilution, that bleeding arrests the convulsions. The remaining conditions named, anaemia of the brain and serous effusion in the meninges, are possible effects of arterial tension, and their influence cannot be separately estimated. The same may be said of the

minute capillary haemorrhages supposed by Dr. Mahomed to be the immediate cause of the convulsions; if they are the proximate determining lesion, they are themselves the effect of extreme high pressure in the arteries. As regards the arterial tension, it is well known that it is raised in almost all diseases of the kidney; and it has always, according to my observation, become extremely high when uraemic convulsions are impending. Convulsions, moreover, are sometimes associated with inordinate arterial tension when there is no disease of the kidneys. Finally, in whatever way venesection acts as a remedy, it certainly lowers the pressure in the arteries. (I must here add, in order to avoid misunderstanding, that convulsions may occur when the arterial system is in the very opposite condition to that of tension, and that in epileptics the pulse is often—usually I should say—remarkably soft, short, and weak.)

But granting that extreme arterial tension is an invariable antecedent of uraemic convulsion, and therefore fulfilling the condition of a cause, this does not explain how the convulsions are produced. It is not yet definitely settled whether the obstruction to the passage of the contaminated blood has its primary seat in the capillaries or in the arterioles. If in the capillaries, then, notwithstanding the contraction of the arteries, which would on this hypothesis be protective, these minute vessels must be exposed to the distending influence of the high pressure in the arterial system, and capillary haemorrhages or serous effusion will be a probable consequence. It has again been supposed that the forcible injection of the brain might so far expand it as to give rise to pressure on the venous sinuses and outlets, which would dam back the blood and cause a degree of stasis in the cerebral circulation. If, on the other hand, the stopcock theory of Dr. Johnson is true, and the primary obstruction is due to the contraction of the arteries, the supply of blood will be limited, and the pressure in the capillaries may be below that of the normal condition. The former hypothesis is, in my opinion, the more consistent with all the facts.

But it is time to describe the *indication for venesection* under consideration, and one more different from the flushed face and full bounding pulse, traditionally supposed to constitute this, could scarcely be imagined. The face is pale; when the fingers are placed lightly on the radial artery pulsation is hardly felt, and the vessel is usually small, though it may be large. The first impression is that the pulse is weak. When pressure, however, is made, so as to test the force of the current of blood, the pulsation is not easily extinguished, and the firmer the pressure the stronger the beat seems to be. At the same time the artery is full between the beats, and can be rolled under the finger like another tendon, or

like a piece of whipcord. The heart will be usually found to be hypertrophied, and more or less dilated, and very commonly there is reduplication of the first sound over the inter-ventricular septum near the apex, due, as Dr. Sibson demonstrated, to want of synchronism in the systole of the two ventricles. When with these evidences of high arterial tension convulsions supervene, the most prompt and certain remedy is venesection.

Chronic renal disease.—Two cases have already been briefly related in which bleeding was of marked service in uraemic convulsions. The disease in both was acute scarlatinal nephritis, and an illustration of its utility in chronic renal disease may be added. The patient was a lady, to whom I was called by Mr. Cripps Lawrence. She was, though comparatively young, the subject of contracted granular disease of the kidney, established apparently in previous pregnancies, and was again pregnant. She was suffering from vomiting, pain in the head, and confusion of the mental faculties, which had rapidly increased, and at the time of my visit she was in a state of stupor. The pulse was extremely small, long, and hard. It was evident that convulsions were impending, and Mr. Lawrence had already warned the husband of this. The warning was repeated, a nurse was set to watch the patient, and it was agreed to endeavour to avert the attack by purgatives and bromide, but that should convulsions come on venesection was to be performed at once, and then premature labour was to be induced. Within a few days the apprehended attack came on. The patient was bled, the convulsions were arrested, and a miscarriage was then brought about. The patient made a good recovery, and regained, and retained for some years, apparent health, under the judicious management of Mr. Lawrence. After various complications she ultimately died from cerebral haemorrhage, and a post-mortem examination confirmed the diagnosis.

Whenever in kidney disease the symptoms of uraemic poisoning show themselves, vomiting, headache, confusion, stupor, muscular twitchings, etc., which experience has taught us lead up to convulsions, we almost always find the signs of undue arterial tension described, and we may confidently anticipate that venesection will at the same time diminish the pressure and relieve the symptoms. I have in several cases both of contracted granular and large white kidney, when cure was hopeless, diminished the suffering of the last few weeks of life, and changed the mode of death, by small bleedings, when the headache became intolerable and convulsions were impending.

An example may be related in which bleeding was several times practised.

The patient, a young man, aged twenty, was first admitted into St. Mary's Hospital in August, 1879, for a slight attack of diphtheria, which, however, set in with a convulsion. As he recovered it was found that he was the subject of chronic albuminuria, which explained the unusual mode of onset of the diphtheria. He recovered from the diphtheria and left the hospital, but returned on November 16, exhibiting all the marks of advanced renal disease and complaining of loss of sight. On ophthalmoscopic examination severe albuminuric retinitis was seen to be present, to which the impairment of vision, already serious, was due. The pulse exhibited all the characters of extreme arterial tension: it was long, hard, and incompressible, the artery being full between the beats, and feeling like a thick cord as it rolled under the finger. The boy was extremely pale and rather weak, but he was on the verge of uraemic complications; and on this account, and in the hope of saving the eyes from further damage, venesection was ordered to eight ounces on November 19. The form of disease affecting the kidneys was considered to be large white kidney from acute desquamative nephritis in process of contraction, and we were not altogether without hope that as bleeding is sometimes promptly curative in the early stage, it might be of service even then. The patient was relieved, and the eyes certainly did not seem to get worse as fast as before, if, indeed, they did not improve; the pulse became larger, softer, and shorter. There was no perceptible difference, however, in the state of the urine, and the symptoms gradually returned, in spite of the usual treatment. On December 17 he was apparently moribund, excessively pale, scarcely conscious, with headache, muscular twitchings, and all the signs of impending convulsions. The pulse was small and long, though not very strong as the heart was acting feebly, the artery was full between the beats and could be rolled under the finger. It seemed as if the loss of an ounce of blood would prove fatal, but as he was clearly about to die and no other treatment offered him a chance; he was bled to eight ounces. The effect was remarkable. He began at once to improve, and so far rallied as to get up daily and go about the ward and to enjoy his food. The tension, however, which was markedly lowered, gradually increased, but we hesitated to take away more blood from a patient who was so extremely anaemic. As we declined the task copious epistaxis came to the patient's relief. On February 11, after premonitory symptoms for some days, convulsions again came on, for the relief of which my resident, Mr. Havell, at once bled to twelve ounces, and as uraemic symptoms were still very severe, and the arterial tension very high, on February 14 he was further bled to eight ounces.

After this the confusion and stupor disappeared and the patient became quite clear in his mind; but he died on February 16, remaining rational to the last. On post-mortem examination the diagnosis of large white kidney becoming granular was confirmed.

Convulsions.—But high intra-arterial pressure may exist independently of kidney disease, and may give rise to convulsions when it reaches a certain degree of intensity. In 1877 I reported to the Clinical Society a case illustrating this, in which venesection was attended with remarkable success. It may be reproduced here, and is as follows:—

The patient came to my consulting-room on Wednesday, July 25, 1877, saying that on the previous Monday morning he had an apoplectic attack. All that he knew about it was that, on coming to himself, at about 6 a.m., he found the house had been roused and a medical friend, Dr. Gaven, sent for. He pointed out a general discolouration on the forehead and chest, which was found to be due to innumerable minute capillary extravasations, and I inferred, what turned out to be the case on inquiry from his wife later, that there had been convulsions. He said he had had a little headache after the attack, but that he then felt perfectly well, and he laughed at the serious view taken of the case. While he was talking with me I observed several momentary suspensions of consciousness, during which he lost the thread of his conversation or ceased to hear what I was saying. I had seen this gentleman before, and knew the main facts of his medical history. He was about forty-two years of age, inclined to be stout, and slightly florid. He was of an excitable disposition, and intellectual in his pursuits. He had spent much time abroad; had lived freely and heedlessly, but had not been given to excesses; had had syphilis when young; had taken much mercury and iodide of potassium, and the last manifestation, which had been rupial ulceration, of which he bore many scars, had taken place eighteen years ago. Exactly twelve months before he had had a carriage accident, in which he received a very severe concussion of the brain, and was supposed to have sustained fracture of the base of the skull. He had been unfit for professional work for three months, and on his return to it did very little for some time, and was extremely careful in the matter of diet and drink. Little by little he had increased his work and thrown off the restraints imposed upon him, and had resumed his old convivial habits. He acknowledged that he was more irritable than before the fall, but considered that no other ill effect remained, except that he had lost the senses of smell and taste. After hearing the patient's account of the attack

and making the observations mentioned I sent him home, ordering him aperient pills and small doses of bichloride of mercury.

Next morning I was called to him, and reached his bedside between 7 and 7.30 a.m. I found there Dr. Gaven, and learnt that our patient had had a succession of violent convulsions from about 3 a.m., at intervals of about fifteen minutes and had been unconscious the whole of that time. He was lying on his back unconscious, tossing restlessly, throwing about his arms, and groaning loudly. The face was expressive of suffering, the eyes closed and held firmly shut when the attempt was made to open them, the pupils equal and of moderate size. It was noticed that the left arm was moved less than the right, but on careful watching it was apparent that the reason for this was some injury to the muscles about the shoulder-joint, and there was no evidence of hemiplegia or paralysis of any kind. The heart was beating violently, but the sounds were normal. The pulse was rather small and weak, but long, and the artery was full between the beats. When an attack of convulsions was coming on the groaning ceased, and the limbs were at rest. The eyes were then opened widely, their look and the expression of the face being for a moment almost natural, but the pupils quickly dilated, the cornea was turned up under the eyelid, the face became distorted, and there came a long and fearful groan, or almost scream, at the end of which the entire body and all the limbs were convulsed with violent synchronous jerks. The face became livid, the pulse imperceptible, and the heart-beat weak, and it seemed as if life were extinct. This would, I think, really have been the case had it not been for Dr. Gaven, who at the critical moment of each attack got a spoon between the teeth, depressed the tongue, and started respiration artificially by pressure on the chest till the respiratory movements began to be re-established.

While we were discussing and deciding upon the line of treatment two attacks occurred, notwithstanding administration of chloroform. Mustard poultices had already been applied to the calves and to the nucha. A dose of calomel was placed on the tongue. An enema was ordered, and then bromide of ammonium, or potassium with chloral, was to be injected into the rectum. Any effects to be hoped for from these measures, however, would require time, and every hour was full of peril. We consequently decided to bleed the patient. This was done by Dr. Gaven to at least thirty ounces. The pulse did not falter during the flow of blood, but became larger and softer. There was no other apparent effect at the moment. Another convulsion came on, the third I witnessed, almost before the bandage could be adjusted. It was as violent as any of the others, but it was the last. The convulsions from

this time ceased entirely. In the afternoon the patient had regained consciousness, but could not speak. Next day he talked very well, but his ideas were rather scattered. In another day or two his intellect was quite clear, and, except that he felt very stiff everywhere, and had pain in the left shoulder, to the muscles of which some injury had evidently been done by the convulsions, he declared himself to be quite well. Bromide of potassium was given, and Dr. Gaven carefully watched the progress of the case. For some time there was much jerking of the limbs during sleep, and once or twice some apprehension was excited by pain in the head, or excitement of manner, but there was gradual improvement, and very soon the patient was able to do a little work. He recovered completely, and is now perfectly well. (He was still well twenty years after the venesection.—Ed.)

Another example of successful bleeding is worth relating, and the more so as the case did not come under my observation till afterwards. The patient was a young lady, aged about 20, who was brought to me in October 1879, with a letter from Dr. Newington of Wheatley, from which the following is abstracted :—She had enjoyed good health, but suffered from constipation and piles, took plenty of exercise, was abstemious in diet, and took no stimulants. The catamenia had been absent for some months, and while abroad during the summer she had suffered from fullness in the head and disinclination for society, and when she walked up hill her face became purple, and she felt as if about to have a fit. These sensations had increased to an extreme degree ; the conjunctivae were injected, and the pulse full and throbbing ; the symptoms were so urgent that Dr. Newington at once bled her on September 21 to thirty ounces. She lay down for an hour, had lunch, and felt so much better that she went to church. Slight discomfort in the head was all that remained, and for this bromide was given, aloes and myrrh at night and Friedrichshaller water in the morning being also ordered. In two or three days she seemed to be quite well ; and she so remained till October 14, when Dr. Newington was again called to the patient on account of a similar but milder attack. Menstruation, however, came to her relief. I saw this young lady at the end of October ; and although she then considered herself perfectly well, there was still high tension in the pulse, and there could be no doubt that it had risen to the point of danger, when she had the fullness in the head and other symptoms. In my opinion, indeed, the bleeding averted a serious catastrophe.

The injurious effects of high pressure in the arterial system are not confined to the production of convulsions. These, indeed,

are rare and exceptional, while innumerable lives are shortened by it in other ways. Even moderate excess of tension in the long run produces disease of the vessels, or of the heart, or of both; and it was in such a condition of the circulation, which was accurately recognized by the more discriminating of our forefathers, that the "spring and fall bleeding," which was so common, would really be of service. I venture, indeed, to affirm that many lives might be prolonged, and many attacks of paralysis averted, by a revival of this practice in certain cases, not that no other means are available, but because such patients would submit to an occasional periodical loss of blood, while they will not exercise the habitual self-restraint and abstinence required to carry out a plan of treatment which must extend over months and years.

I am particularly anxious that it should not be supposed that bleeding is indiscriminately recommended as a remedy for convulsions. Only when high arterial tension is present should it be employed.

Cerebral conditions.—It was formerly the practice to bleed after severe injuries to the head, especially when symptoms of so-called cerebral irritation came on. I have no doubt there are cases in which it would be of great service, but it is a matter in which I have no experience. It might seem at first sight that bleeding ought to be a valuable resource in cerebral haemorrhage; but careful thought would lead to the conclusion that its operation could only be extremely limited, and this is what is found in practice. We should not be so liable now to take the effects of embolism and thrombosis for those of rupture of a vessel, and so do serious harm instead of good; but in a large proportion of cases of cerebral haemorrhage the mischief is done before the patient comes under observation. In ingravescent apoplexy, however, the oozing of blood appears to continue for some time, and as this form of attack is almost invariably fatal, it would be well worth while to try what bleeding could do.

While bleeding comes too late when an artery has already burst in the brain, it might often avert such an event, and when the indications of excessive arterial tension have been well mastered, and are distinctly recognized in a patient who presents any of the premonitory symptoms of apoplexy, venesection ought to be fearlessly practised. Almost every medical man will have seen cases in which epistaxis has at once brought relief from threatened apoplexy, and it is in our power to afford relief equally efficacious.

It is probable that there are other conditions in which bleeding would be of great service; but it is better to proceed with extreme caution in the reintroduction of this practice. There is a strong

prejudice against it in the public mind, though not, in my experience, so strong that it will be forbidden when it is recommended by a medical man who has confidence in its power to do good; and this makes it important, in the interest of the medical man, that there should be no doubt as to its beneficial effects whenever it is tried. The great service, again, which venesection is capable of rendering in suitable cases is a weighty reason for desiring that its employment should not be retarded by resort to it in cases where its effects are doubtful. The ground gained should be made sure by experience before further advance is attempted.

PNEUMONIA

From *The Practitioner* for January, 1900

PNEUMONIA is a very remarkable disease. Whatever criterion may be applied, it must be looked upon as an inflammation, but there is no other inflammation of an important organ which at all corresponds with it in the suddenness of its onset, the rapidity of its progress, and the abruptness of its termination.

It is, as the Registrar-General's Reports show, responsible for a large number of deaths; but in a considerable proportion of the cases it is, to quote Sir William Gull, rather a mode of dying than a cause of death. Pneumonia, sometimes of a very acute type, may constitute the closing scene in old age, or in debilitated constitutions, or may step in at a late stage of many acute or subacute diseases—in small-pox and the fevers generally, in acute rheumatism, in acute or chronic renal disease; or it may attack an apparently strong and healthy person at any period of life.

Aetiology.—The peculiar character and course of the inflammatory process in pneumonia are explained by the fact that it is due to an infective organism, the pneumococcus. It is open to discussion whether the affection of the lungs stands in a similar relation to the microbe and the febrile process as the rash of scarlet fever, or resembles diphtheria, and perhaps erysipelas, in the sense that the disease is primarily local, the constitutional symptoms being due to a toxin formed in the pneumonic focus. From time to time the pneumococcus seems to acquire a degree of virulence which renders the disease contagious, and there is an epidemic of pneumonia on a small scale. For the most part, however, some contributory cause can be traced, which may be the debilitating influence of the diseases in the course of which pneumonia is liable to occur; the most common antecedent, however, is exposure to cold, and the fact that the disease is the work of a microbe must not make us oblivious of the important part played by chill in the causation of pneumonia. It is assumed that the temporary lowering of the resistance to microbic invasion affords the diplococcus a chance. It is not necessary to assume that the diplococci are ubiquitous, and lying in wait everywhere for such opportunity. Apparently they are carried about in the mouth and pharynx,

and it is probable that unhealthy secretions here afford them a suitable nidus.

Symptoms.—Pneumonia runs a course which is remarkably consistent. The onset is abrupt, and is usually marked by feelings of chilliness, which may be attended with actual shivering, occasionally amounting to rigor. There is speedy reaction from this, and the face becomes flushed, the skin hot and burning to the touch, and the respirations frequent. The pungent heat of the skin is remarkable, and is a noteworthy point in the diagnosis. Cough comes on early, and soon becomes frequent and trying. While it varies greatly in severity, and in the degree of attendant distress and pain, it is generally harsh in character. The expectoration, scanty at first and rarely profuse at any period of the disease, is extremely tenacious, and almost always tinged with altered blood. There may be streaks of actual blood, but in the characteristic sputum the blood colour is diffused and modified so as to give the well-known pale or dull rusty appearance. When collected in a vessel this rusty sputum is seen to be permeated by minute air bubbles, and its tenacity is such that it does not flow when the vessel is tilted, but clings to it even when it is reversed. The expectoration may be dark in colour, liquid and copious, when it has been likened to prune-juice. This is a very ominous indication.

The temperature, which will have begun to rise during the preliminary chill, quickly reaches its maximum, which will be 103° or 104°, or occasionally 105°, and the diurnal variations are slight. The pulse becomes frequent, and it has the character which has been described as full and bounding, i.e. the arterioles are relaxed, the radial artery is large, the ictus sharp, sudden, and forcible, while the pressure, from the absence of resistance in the arterioles and capillaries, falls very rapidly, and the beat is therefore short. Dicrotism is usually conspicuous in the sphygmographic trace and can generally be detected by the finger. The blood, so to speak, is allowed to shoot through the capillaries, and when the radial artery is obliterated by pressure of the finger, pulsation can be felt on the distal side which has come round through the palmar arch.

The urine is high coloured, and liable to throw down lithates on standing; it may contain a little albumen, and it is always remarkably deficient in chlorides, which are said to be withdrawn from the blood by the exudation in the lungs. This explanation, however, can scarcely be accepted as satisfactory from a quantitative point of view, and the same may be said of the diminished supply of chlorides in the food. It seems probable that the tissues generally may have a heightened affinity for the sodium chloride and retain it during the febrile state.

Pain is usually experienced early on the affected side from concomitant pleurisy, and the surface temperature of this side of the chest is higher, the surface thermometer rising also more quickly.

When the respiration becomes so far embarrassed that dyspnoea may be said to exist, the alae nasi will be in motion, inspiration will be attended with visible effort, expiration being very short and sudden.

Physical signs.—The diagnosis is frequently, perhaps usually made independently of physical signs, and often before they become distinct and characteristic. When fully developed, these are: dullness on percussion, with a sense of resistance; tubular breathing, with a marked and peculiar expiratory whiff, associated probably with the short and rapid expiration; bronchophony and increased vocal fremitus. These signs indicate complete solidification of the part of the lung affected, by exudation into the air vesicles. They may be preceded by fine crepitation over the area affected; and the solidified block, and especially its advancing edge, may be fringed by this fine crepitation. Fine crepitation was at one time regarded as an essential part of the diagnostic physical signs. In many cases, however, crepitation may never be detected.

A friction rub can often be heard over the seat of the painful stitch in the side.

The base of one or other lung is the part most frequently attacked, the right more often than the left. Double pneumonia, in which first one base and then, at a short interval, the other is affected, is not uncommon.

While pneumonia mostly affects the base of the lungs, the apex may be the part attacked; and I have seen a few cases, always associated with influenza, in which the middle lobe of the right lung has undergone complete consolidation, both apex and base escaping.

In apex pneumonia the development of physical signs may take place very late, when the dullness and tubular breathing may supervene with great rapidity, the tubular breathing being very pronounced and obtrusive. Remarkable skodaic resonance may be present on percussion below the clavicle when the posterior part of the apex is consolidated. The crisis may follow very closely upon the consolidation.

The consolidation of the lungs advances rapidly, and it is not uncommon to find it extending up to the spine of the scapula on one side and half-way up the dorsum of this bone on the other, or the back of the lung may be solid from base to apex. It spreads for a varying distance forwards on the lateral aspect of the chest, not commonly round to the front. Why it should stop at all, and

what determines its arrest, is not clear; but pneumonia is not often fatal simply from the extent of lung involved.

As the disease progresses the face loses its flush and becomes pale, and in very severe cases tending to a fatal termination, livid, the skin remaining hot and burning. The eyes will have an anxious look, the countenance an expression of distress, especially when there is dyspnoea. The rapidity of the respiration is maintained or greatly accelerated. The pulse loses force and volume, and may become more rapid; and the right auricle and ventricle will be found to be distended. The tongue is coated, and perhaps dry and brown, or red and angry-looking. Sleeplessness is a distressing feature, and it may be almost absolute. Delirium is common, and sometimes violent, and patients suffering from pneumonia have been known to commit suicide.

The crisis.—Fortunately pneumonia is a disease of short duration. On the sixth or seventh day, sometimes earlier, a crisis occurs, marked by a rapid drop of the temperature and subsidence of all the symptoms. A sound, refreshing sleep is one of the usual incidents of the critical improvement, and there may be perspiration.

The crisis may be delayed till the seventh or eighth day, and apparently the invasion of the second lung may be responsible for such delay. Should it not have come on the ninth day, it may be concluded that there is some underlying cause, such as tubercle or septicaemia.

If an examination of the chest is made immediately after the crisis, the consolidation of the lungs will be found unchanged. The crisis, therefore, is not due to any improvement in the condition of the lungs, but this soon follows. Air begins to penetrate the bronchiae and air vesicles, and gives rise to the coarse redux crepitation by the minute bubbles which form and burst in the liquefying exudation. This exudation must be largely absorbed by the capillaries and lymphatics of the lung, since comparatively little of it is expectorated. All trace of it is eventually removed, and the lung becomes perfectly clear. "Unabsorbed pneumonic deposits" have been spoken of, but are practically unknown as post-mortem appearances.

Varieties.—While the symptoms are such as have been described, and the course of the attack and mode of termination may be the same, the consolidation may in some cases never be complete, and with the dullness, bronchial breathing, and bronchophony there is throughout loud, harsh, coarse crepitation.

The expectoration is usually more copious in such cases.

Pneumonia intercurrent in the course of other acute diseases has not apparently the sudden onset which is seen in an ordinary

attack. It may determine a rise of temperature, and the frequency of the respiration and probably of the pulse will be increased, but it is usually recognizable by physical signs before its existence is made known by obvious symptoms.

In enteric fever it is usually towards the end of the attack that pneumonia supervenes, and it is not always easy to distinguish between true pneumonia as a complication and the consolidation from passive congestion which has received the name of hypostatic pneumonia. When, however, the case has been carefully watched no difficulty can arise, as the stasis of blood in the back of the lung comes on slowly, and the imperfect entry of air, the appearance and spread of crepitation, and the gradual impairment of resonance can be followed from day to day. When the consolidation is detected elsewhere than at the base and posterior part of the lung it is pneumonic.

In rare instances pneumonia supervenes during the early stage of convalescence from typhoid fever.

Pneumonia associated with influenza may form part of the attack or may come on after the stormy symptoms attending the onset have subsided, or may follow the actual influenza at varying intervals, not unfrequently appearing to be due to exposure or exertion during convalescence.

It is usually characterized by asthenia, is always serious, and in old people dangerous. It is liable to be followed by empyema. The apex or middle lobe may be the part attacked, or the dullness may be patchy and shifting. As is the case with asthenic forms of pneumonia generally, the onset is insidious and the crisis late and less distinctly marked. Herpes labialis is rarely present.

Catarrhal pneumonia.—The catarrhal form of pneumonia differs from pneumonia proper in the fact that individual lobules are affected, and not the lung substance *en masse*. Perhaps lobular pneumonia is a better name for it. The lobules, in greater or less number, are choked by exudation and rendered impervious to air, and the consolidated lobules may be so numerous and so closely packed that a considerable mass of lung may be solidified.

This is the form of pneumonia which so commonly complicates measles and pertussis in children, and in later life it may occur in persons subject to bronchitis, and may supervene upon bronchitis. No definite line, indeed, can be drawn between catarrh of the finer bronchial tubes and catarrhal pneumonia.

The onset is usually insidious, and the transition from bronchial catarrh, say in a child, is marked by a rise of temperature, and by increased frequency of the pulse, and especially of the respiration. It is not uncommon to find the respirations sixty in a minute, the

alae nasi in conspicuous play, and the larynx travelling up and down. There may be inspiratory retraction of intercostal spaces and, in children, falling in of the lower ribs from atmospheric pressure. The chief point of difference between lobular and lobar pneumonia is the predominance of respiratory over toxic symptoms in the former.

The dullness over the affected lung is less absolute and less uniform. Air is not completely excluded, and sibilus and rhonchus are usually heard as well as tubular breathing.

Prognosis.—A forecast of the probable course and issue of an attack of pneumonia can usually be made before the development of physical signs. In this the age and antecedent condition of the patient will form an important element. Old age, debility, obesity, alcoholic habits (especially when there is already damage to the liver), diabetes, kidney disease, valvular or structural disease of the heart, pre-existing disease of the lungs themselves, are in different degrees serious. A distended condition of the abdomen, from whatever cause, adds gravity to the case, whether it is due to paresis of the intestinal muscular walls and indicative of asthenia, or is the result of derangement of the digestive organs, and diarrhoea may be a serious complication.

The aspect of the patient, the frequency and character of the pulse, and the degree of respiratory disturbance and distress will be most important indications, earlier and more important than the physical signs ; and the changes from day to day, and almost from hour to hour, are full of significance, the pulse and respiration being counted and registered at each visit, and perhaps in the intervals, by the nurse. Delirium, especially if it comes on early, is always serious. The temperature takes a secondary place in prognosis.

The mode of onset has its significance. When sharp and attended with rigor we can reckon on the crisis with greater confidence than when it has been insidious. Herpes labialis, which is justly regarded as a favourable sign, is usually associated with initial rigor, as pointed out by Mr. Hutchinson.

When the consolidation supervenes, its situation, rate of advance and extent will enter into the prognosis, and especially the invasion of the other lung. Pneumonia of the apex is more serious than pneumonia of the base, and more liable to be attended with delirium. When the subject of it is not a child, it is usually of septic, or influenzal, or sometimes of rheumatic origin, or is associated with renal disease or alcoholic habits. Pneumonia localized in the middle lobe of the right lung I have met with only in influenza.

The physical signs will be carefully watched, but the disease rarely proves fatal simply from the extent of lung involved.

Albuminuria coming on during the attack, if considerable in amount, is serious. Jaundice still more so.

When the consolidation begins to break up, and entry of air and redux crepitation are heard without the occurrence of a crisis, there is extreme danger. There appears to be rapid absorption of the exudate, which acts as a toxin, giving rise to delirium or to hyperpyrexia.

Treatment.—Pneumonia has been from time immemorial the battle-ground of therapeutics. Every theory and every line of practice has in turn appealed to the results obtained in pneumonia as proof of its truth and of its success. It was in pneumonia that the efficacy of venesection was supposed to be unquestioned, and it was by the application of the numerical method to pneumonia, and the consequent overthrow of this superstition, that Hughes Bennett banished general bleeding from medical practice in this country. So long as the clinical history of pneumonia was not observed apart from interference, and disease of all kinds was looked upon as a morbid entity which had to be expelled by remedies, it is easily understood that the crisis, with the striking favourable change which then took place, was put down to the treatment employed—bleeding, stimulants, calomel and opium, mercury, antimony, or whatever happened to coincide with the improvement.

This is a lesson we may still take to heart. Pneumonia being a disease of short duration, with for the most part an assured critical termination in a few days, we can afford to watch and wait. The temperature is not a source of danger, either from the height to which it rises or from the time it will remain high. Nothing is to be gained, therefore, by violent efforts to reduce it, either by the external application of cold or by antipyretic drugs. Cold or tepid sponging will be a comfort to the patient, and may soothe and facilitate sleep; beyond this it is not necessary to go. Antipyretics, such as antifebrin, antipyrin, phenacetin, given continuously are not only useless but injurious. A single dose at the outset has, in the experience of competent observers, seemed to cut short an attack, and an occasional dose early in the disease may relieve headache and procure sleep, and is, therefore permissible; but the systematic administration of any of these drugs depresses the heart and impairs the resistance to the debilitating effects of the disease—possibly, indeed, interferes with the cycle of processes by which the crisis is reached.

In the conduct of a case the line of treatment must be determined by the antecedent condition of the patient, and by the front he presents to the attack; and the attention of the medical

man must be directed mainly to the early recognition of any functional derangement or complication, or any indication of failing power.

Precise directions as to food would here be out of place. The nourishment will, of course, be liquid and adjusted to the digestive capacity and general condition of the patient, any idiosyncrasy being respected.

Stimulants are rarely necessary, often useful. The rule should be to reserve them till they are indicated by a frequent, small, short and weak, or faltering pulse, a dry tongue, restlessness, or delirium. If the patient sleeps after a little brandy or champagne, or if the pulse becomes less frequent and larger, it has done good. In some exceptional cases a considerable amount of brandy may be required, perhaps ten to twelve ounces of spirit, in the twenty-four hours, and sometimes brandy given in champagne seems to have a better effect than either alone.

When *sleeplessness* is persistent and distressing there need be no hesitation in giving morphia hypodermically, beginning with a small dose. Morphia is all the more useful if there is delirium.

In a large proportion of cases it cannot be said that the regular administration of *medicine* of any kind is necessary, but it is usually a comfort to the patient and friends to have the feeling that something is being done. Simple salines, acetate or citrate of ammonia and citrate of potash, allay thirst and exercise some influence on the cough. They are more refreshing when in the form of an effervescing mixture. Carbonate of ammonia and bark may be added when indicated by a flagging pulse, or one or two grains of quinine may be given in the form of tabloid or pill, or dissolved in the acid of the effervescing mixture.

Strychnine is often of very definite service when the cardio-vascular system is manifesting an asthenic tendency. It is best given hypodermically, in doses of $\frac{1}{60}$ gr. to $\frac{1}{30}$ gr., at intervals of from twelve to four or three hours, and it is important to note that it may be required early.

Digitalis has been elevated almost to the level of a specific in the treatment of pneumonia, and it may be extremely useful on occasion, especially as digitalin, with strychnine hypodermically; but given at all periods of the disease, and in large doses, it is of very doubtful value.

The inhalation of *oxygen* is attended with striking, but usually fugitive, apparent benefit when there is lividity of the countenance and blueness of the lips, and it is possible that it may, with strychnine and stimulants, carry a patient through a very dangerous phase of an attack.

Dr. Lees advocates the application of an *ice-bag* to the chest over the affected lung, and external cold has been found to relieve the pain of the associated pleurisy, and is often said by the patient to be grateful. Cases have been published in which the ice-bag has seemed to shorten the attack; but, for reasons already given, nothing can be more difficult than to form a definite opinion on this point, and it is not easy to see how the course of the disease, of which the inflammation of the lung is a local expression, can be so influenced. A large body of statistics would be needed to establish the beneficial effects.

Poultices were until recently almost universally applied, the first one or two usually containing mustard so as to redden the skin. The practice has its justification in the relief of pleuritic pain and of the cough, but a large jacket poultice constantly applied soon becomes oppressive, and when changed every three hours it is a cause of fatigue and distressing exhaustion. All that is valuable in the use of poultices may be retained without the disadvantages attending their too assiduous employment by the application of one for three hours twice in the twenty-four hours.

Legends are still in circulation of life having been saved in pneumonia by a gigantic blister, and it is still possible that the medical attendant may be pressed to apply a blister.

Venesection has rightly been banished from the treatment of pneumonia, but in one particular complication, very rarely met with, it may be of the greatest service. This is when early in the attack the invasion of the lung is so rapid that the right ventricle is unable to cope with the sudden resistance in the pulmonary circulation, and is paralysed by over-distension. The patient will be cyanosed, unable to speak, and scarcely able to breathe or cough; he will be sitting up in bed with his legs over the edge unless prevented, supporting himself on his hands, the veins of the neck and temples turgid, the eyes staring, the expression agonized, and beads of sweat standing on the face and forehead. The pulse will be small and short, probably scarcely perceptible, while the heart will be found beating violently. It is scarcely possible to define the heart by percussion, but the right cavities are enormously distended, and the contrast between the cardiac impulse and the pulse shows that the blood is dammed back in the lungs so that it reaches the left ventricle in very inadequate amount. Under such circumstances the relief by bleeding is most striking. The pulse improves as the blood flows, the breathing is relieved, and when sixteen to twenty ounces have been withdrawn all the distressing symptoms will have disappeared.

The same results are not to be expected later, when asthenia

has become a prominent feature in the symptoms, although, of course, there is dilatation of the right heart.

It has been already stated that *delirium* is common in the course of pneumonia. It usually subsides at the crisis, especially when with this there is a long, quiet sleep. But it may persist and become more violent, or violent delirium may set in after the crisis. It is probably of toxic origin, the source of which may be the exudate absorbed into the blood, or it has been attributed to asthenia. The remedial measures are nourishment and stimulants, with strychnine and digitalin hypodermically.

A CLINICAL LECTURE ON A CASE OF PNEUMONIA, WITH ABORTIVE CRISIS AND PREMATURE RESOLUTION, FOLLOWED BY SUPPURATION OF THE BRONCHIAL GLANDS

Delivered at St. Mary's Hospital

British Medical Journal, 1898. Vol. I

The patient, a strong healthy boy of 12, was taken ill on the evening of December 2, 1897, when he was sick and felt chilly. There was no distinct rigor. Five days before he had played in a football match, and two days later had been out in a storm to watch the wrecking of the Broadstairs pier. He had a slight cold and cough, as also had other boys in the school, but there was nothing suggestive of influenza in the symptoms. The attack speedily assumed the characteristic features of pneumonia, and when I saw him on the 5th, the temperature was 105°, the pulse 120, the respiration 48. There was a harsh cough, without expectoration, and pain in the right side, but in the abdomen rather than in the chest. The physical signs developed slowly, and it was only on the morning of this day that Dr. Raven had recognized indications of commencing consolidation of the right base. These were more distinct at the time of my visit, and distant tubular breathing was also to be heard above the spine of the scapula. We were agreed that the case was one of pneumonia, that the attack was severe, but that there were no special grounds for anxiety. A high temperature in pneumonia is not necessarily a very serious matter, especially at the age of 12. The respiration, it is true, was very rapid, and we should like to have had a good crop of herpes on the lips, but we saw no reason to apprehend a fatal issue. We prognosticated a crisis on about the seventh day, and recovery in due course. On the 7th Dr. Raven reported that the right lung was consolidated from top to bottom posteriorly, the temperature and symptoms generally remaining much the same. So far there was nothing unusual in the course of the attack.

Abortive Crisis.—On the 8th, however, instead of the crisis we were expecting, with a rapid fall of temperature to normal or subnormal and a general amelioration in the symptoms, the temperature only fell to 103°, and the boy, instead of being better, was obviously

worse in all respects; he became delirious, and the pulse went up to 144, and the respirations to between 50 and 60.

I was therefore asked to see the patient again, and reached his bedside at Broadstairs about 10 a.m. on the 9th. The boy was lying on his left side, which was his usual position throughout, and although naturally very alert, he took no notice of what was passing in the room; from time to time he made some rambling remark, but when roused he knew me, and expressed surprise at seeing me. There was a deep crimson circumscribed flush on each cheek, the pulse varied from 130 to 140 and upwards, and he was breathing at the rate of 64 times a minute. The temperature had just been taken, and was found to be 103°.

Premature Resolution.—On proceeding to examine the boy, instead of the dullness on percussion which I expected to find, there was everywhere resonance. At no point was there a trace of the consolidation which less than forty-eight hours before had involved almost the entire lung. On auscultation, again, air penetrated freely every part of the lung, and neither rhonchus nor sibilus nor redux crepitations could be detected anywhere. The inspiratory murmur was harsh, and the expiratory murmur was distinct. Dr. Raven confirmed these observations, which contrasted in the strongest manner with what he had found only the day before.

Nothing could be more grave than the state of things thus revealed. We should expect so striking a deviation from the usual course of events in pneumonia to be serious on various *à priori* grounds, and as a matter of experience the patient who is the subject of this lecture is the only one whom I have known to survive. Happily such cases are rare. As you are all well aware, the usual termination of pneumonia is by crisis; the temperature falls, the pulse quiets down, the respiration becomes less frequent, the tongue cleans, and a tranquil sleep brings to an end the delirium or stupor, the muscular tremor or twitching, or other evidence of nervous prostration or disturbance, which may have supervened; but the crisis relates to symptoms only, not at all to physical signs, and while the patient may have all the appearance of convalescence, dullness, tubular breathing, and bronchophony are found to be as distinct as ever, and a week or ten days will be occupied in removing the exudation from the air cells. Now whatever view we may take of the intimate changes which bring about the crisis, whether we attribute it to the death of the pneumococcus or to the formation of some antitoxin, or to the completion of some process of elimination, the crisis with which we are familiar is clearly Nature's method of bringing the disease to an end, and with

each day of delay in its appearance anxiety as to the result increases. When it is postponed beyond the ninth day it may usually be concluded that something underlies the pneumonia, such as tubercle or septicaemia; but even more serious than delay of the crisis is an abortive attempt. Nature has made her effort and failed, and nothing more is to be expected of the constitution; but there was another obvious source of danger. Two days previously the air vesicles of a great part of the lung had been choked with exudation which had now entirely disappeared; it had certainly not been expectorated or brought up or swallowed; it could only have been absorbed, and from its character there was every reason to fear that it would play the part of a poison in the blood. The patient indeed was evidently under the influence of a poison.

It is not easy to understand such rapid absorption, and I make no attempt to explain it. I cannot help thinking, however, that the deep and frequent respiration is a factor in the causation. In the only other case of the kind in which I have had the opportunity of minute and continuous observation there was frequent and deep breathing and incessant loud talking, at first sensible and coherent, gradually becoming incoherent. In this case, which was watched minutely, the clearing up of the consolidation took place from below upwards, redux crepitations and entry of air being first heard at the extreme base, first of one lung, then of the other, the pneumonia having been double. Both lungs were clear in less than thirty-six hours.

Hyperpyrexia.—The boy, then, lay in the condition of torpor with muttering delirium, just described, and a point, which at once struck us, and to which I call your attention, was that, while the degree of fever explained the frequency of the pulse, there was nothing whatever in the state of the lung or heart to explain the inordinate frequency of the breathing, which was at the rate of 60 to 64 respirations to the minute. Such a departure from the normal relation between the pulse and respiration added greatly to the gravity of the symptoms. It could only be due to an effect of some poison on the nervous system, or, as was suggested by the sequel and by another case which I have seen, to some irritation at the root of the lung. After the examination, as the temperature seemed to be rising, it was again taken and found to be 103·8°; half an hour later it was 104·8,° upon which the boy was twice freely sponged with cold water, and an ice-bag was applied to the head. This had little effect, since in another hour the temperature had further risen to 105·6.° Upon this the patient was enveloped in a dripping sheet, which was so disposed as to favour abstraction

of heat from every part of the surface, and the moisture was continually renewed by droppings of ice-cold water from a sponge. The temperature was taken in the mouth every few minutes, and it was fully twenty-five minutes before any impression at all was made on the heat. Of this the first indication was paling of the flush on the cheeks, and when this became very decided the wet sheet was withdrawn, the boy, still naked, being covered by a single blanket. He enjoyed the cold application very much, and became quite sensible. The temperature fell to about 103° for a time, but the wet sheet was again required three hours later for a period of a quarter of an hour. Five grains of quinine were given after the first wet pack, and again later in the day.

There can be little doubt that the wet sheet saved the boy's life. He was getting visibly worse in every respect from hour to hour, and was apparently drifting rapidly into hyperpyrexia.

From this time the temperature gradually fell for some days, always, however, touching 101° at some period in the twenty-four hours, and there were at times oscillations which demanded a dose of quinine. The pulse remained frequent, and the respirations maintained a great relative rapidity of about 40. The intellect was clear, and the patient took his food well. Cough persisted, and the lungs were frequently examined with a view to the detection of any morbid condition which might have been overlooked, or of any new complication which might arise, or of anything which might explain the rapid breathing and persistent temperature. During the afternoon of the 10th a very scattered, coarse crepitation was heard for a moment over the upper part of the right lung anteriorly, but it was dismissed by an act of coughing, and from time to time afterwards fugitive signs were detected at different points. We had in mind in particular the possibility of an empyema imprisoned between the base of the lung and the diaphragm, or in the fissure between the lobes of the lung, but no evidence of any such complication presented itself.

Suppuration of Bronchial Glands.— At the end of a week, during which there had been apparently a distinct though slow progress towards convalescence, the temperature rose slightly to 102° and 102·6°, and the cough became more violent and continuous, but without expectoration, beyond such as was excited by the act of coughing itself, and on December 21 I again saw the patient. By this time the paroxysms of coughing were uncontrollable, and would go on for two or three hours without intermission. No linctus gave relief, and inhalations of pine oil and of hot vapour with benzoin afforded no alleviation whatever. The cough was almost like that of whooping-cough, and from time to time provoked

vomiting. An attempt was finally made to quiet it by inhalations of chloroform, but nothing short of complete anaesthesia sufficed to cut short the paroxysms. There was then an interval of quiet sleep, and the cough did not recur for some hours. It was observed that talking, eating, and turning on the right side provoked attacks.

There could be no doubt as to the significance of a cough of such a character; it could only be due to enlarged bronchial glands. Irritation and inflammation of the glands at the root of the lung had obviously been set up by the material absorbed from the air vesicles. The lymphatics had thus been one channel by which the exudation had been removed.

Small doses of iodide of potassium were given, in the hope of reducing the size of the glands, and the cough was quieted as far as possible by an opiate and bromide linctus and by inhalations of vapour with tinct. benzoin and succus conii, or of pine oil, or from time to time of chloroform. On the 23rd a chill, scarcely amounting to a rigor, and a sudden rise of the temperature to 104·2°, announced suppuration. This could scarcely be elsewhere than in one of these enlarged and inflamed bronchial glands, and unmistakable evidence was furnished of pressure upon the root of the lung by imperfect entry of air into the right lung generally, with distant tubular breathing in the first space near the sternum, and slight impairment of resonance in the right interscapular space about the level of the spine of the scapula. On the 24th, when I again saw the patient, it was further found that the heart was considerably displaced to the left. The apex-beat was outside the anterior axillary line, and unduly high. Next day it was as far out as the mid-axillary line, and about the level of the nipple, and the upper line of dullness ran along the second space. These physical signs raised the question of acute general dilatation of the heart and of effusion into the pericardium, but the apex-beat and right ventricle impulse were too definite and the sounds too distinct and too nearly normal for either of these conditions. There was, moreover, no dullness to the right of the sternum, and the heart sounds were scarcely audible outside its right border. The heart was evidently carried bodily to the left, and as there was no fluid in the right pleural cavity, either free or encysted between the diaphragm and the base of the lung, the only assignable cause was swelling of the mediastinum, possibly an abscess there. Corroborative evidence of mediastinal implication was present in conduction of the tracheal breath sounds to the manubrium, especially over its right half, but there was no pressure on the vena cava, the veins of the neck not being full.

The outlook at this stage of the case had again become very

uncertain. There could now be no doubt that suppuration had taken place in the mediastinum. We could be practically certain that it had started in a bronchial gland at the root of the lung, but there was reason to fear that it might have extended into the connective tissue of the mediastinum. Everything depended on the outlet which the matter might make for itself. We hoped it might be into a bronchial tube, but we could not be sure of this, even if the abscess had formed in a gland. A mediastinal abscess properly speaking could only be expected to open into the pleural cavity, and that after a long and trying period of suppurative fever.

Happily on December 25 a small quantity of pus suddenly appeared in the scanty colourless expectoration, which was all that had hitherto been seen, and soon afterwards a single streak of blood. The amount could not have been anything like a drachm, but its escape was followed by a temporary amelioration of the symptoms; for three days the temperature did not exceed 101·4°, and the cough became much less violent and the respirations less frequent. The temperature, however, again rose to 102·6° and had a suppurative character, and there were profuse night sweats. The displacement of the heart was unchanged, so that the menace of perforation of a mediastinal abscess into the pleural cavity was not entirely withdrawn. In view of this contingency, the boy's condition now permitting of the journey to London, he was brought up from Broadstairs by Dr. Raven on December 29. There was now dullness at the right base posteriorly more than half-way up to the angle of the scapula and round the lateral aspects of the chest, with absence of breath sounds, impairment of vocal resonance, and extinction of vocal vibration; there was, in fact, for the first time, fluid in the pleural cavity. This seemed like a preparation for rupture of a mediastinal abscess in this direction, and I rather welcomed the appearance of this fluid, first because I had in a previous case of abscess in the mediastinum known serous effusion, seen to be such when withdrawn by aspiration, to precede the penetration of pus into the cavity; secondly, because I thought the fluid might dilute the pus and diminish the irritation of the pleura which it would set up.

The anticipation based on the appearance of pleural effusion was, however, never realized. A second expectoration of a small quantity of pus took place on December 31, but after this the fever was rather higher, and the oscillations and perspirations more marked, till on January 4 another and more copious expectoration of pus took place. It was not preceded by cough, but came on after a slight effort, and so suddenly that the first outburst could not

be collected. From what I saw I estimated the amount to have been 3 or 4 drachms. Considerable coughing followed the appearance of the pus for the rest of the day.

There had been improvement from the time of the first expectoration of pus; it continued steadily and more rapidly after the final discharge. The temperature speedily fell to the normal level or below it, and the perspirations entirely ceased. The boy got up for the first time on January 8 and left for the country on January 24, not, of course, very strong, but otherwise apparently well. The heart had not regained its normal position, and the second sound was reduplicated, and there was still a zone of dullness round the base of the lung.

The improvement has continued. Dr. Walter Broadbent reported on January 28 the apex-beat to be 1 inch outside the nipple line, and the second sound to be reduplicated at the base. A week later the apex was only ¼ inch outside the nipple, and the aortic and pulmonic second sounds were synchronous. On February 15 the heart had practically regained its normal position in the chest; tracheal breathing was no longer heard over the manubrium, and all that remained at the base of the right lung was a scarcely recognizable impairment of resonance and of respiratory murmur.

INTERLOBAR EMPYEMA

From *The Practitioner* for February, 1905

A FORM of empyema which has not received adequate attention is where an accumulation of pus is imprisoned in the fissure between the lobes of the lung. It receives only a brief mention in Dr. West's excellent treatise on diseases of the lungs, in which it is said to be difficult of diagnosis, and I am not aware that it has received much notice in any work.

I have seen several cases in which I have formed the opinion that this condition existed, but only two in which the opportunity has been afforded of verifying the diagnosis by operation. I did not meet with an example in the post-mortem room during my active connexion with St. Mary's Hospital, but before proving fatal an empyema in the interlobar fissure would probably make its way into the general pleural cavity. Possibly interlobar empyema will be found to be more frequent than is apparent at present, when attention has been called to the condition and it is carefully sought for, as has happened in so many other forms of disease.

In one case in which I came to the conclusion that there was empyema between the upper and lower lobe of the left lung, the pus made its way to the surface above and just outside the mamma, and the patient made a good recovery. In another, on the right side there was sudden purulent effusion into the pleural cavity, and the usual operation was performed. I was not present, and had not the opportunity of watching the case afterwards. In several the pus has burst into a bronchus. It will be seen, on reflection, that this last mode of termination is not an unlikely one. It may almost indeed be inferred that when an empyema is discharged through a bronchus, without the occurrence of pneumothorax, it has been localized in the fissure. As fluid accumulates in the pleural cavity the lung shrinks and retreats before it, first from the removal of the negative pressure, later from the actual pressure of the fluid, till ultimately it is flattened around its root against the mediastinum. It is difficult to see how the fluid could gain access to a bronchus under this condition. Adhesions, however, or solidification might prevent the collapse of

the lung, or the perforation may take place from within outwards, from a cavity, or by a process of ulceration, but the aperture which allowed the exit of the pus would probably permit of entry of air giving rise to pyo-pneumo-thorax.

When the pus is imprisoned between the lobes of the lung, as it accumulates it will tend to split the fissure in the direction of the root of the lung, where it would obtain ready access to the primary divisions of the main bronchus.

One of the cases in which the pus was evacuated by operation I watched with Dr. Hector Mackenzie. It was that of a gentleman nearing the age of 60, tall, muscular, and rather stout. He had had an attack of influenza, attended with some inflammatory affection of an indefinite character of the right lung. He recovered, but some weeks later had a relapse, with return of trouble in the right lung. Very soon the general symptoms pointed to empyema, but there was no such dullness on percussion over the lower part of the chest as to indicate the presence of fluid, and exploratory aspiration, which had been practised at different points during my absence from town, gave negative results. The evidence on which the diagnosis was ultimately arrived at was that the physical signs, slight impairment of resonance and imperfect entry of air, varied from day to day, and, more particularly, that they shifted from the upper to the lower lobe and back from the lower to the upper in a remarkable way. It was inferred that there was probably an empyema between the lobes which pressed upwards or downwards, according to the position in which the patient lay. Careful search was therefore made along the course of the two fissures between the middle and upper and the middle and lower lobes, but no definite dullness could be made out. Dullness was, however, found behind above the spine of the scapula, and from this spot towards the axilla. At length the dullness could be recognized near the apex of the axilla by Dr. Hector Mackenzie and myself, and Mr. Makins was asked to operate. A trocar was passed upwards and inwards, with a slight direction backwards at the point where dullness was present near the apex of the axilla; pus was found, and Mr. Makins resected a piece of rib—no easy matter in this situation—more than half a pint of pus escaped, and a tube was introduced. The progress of the case was extremely good. The drainage tube was extruded with unusual rapidity, and somewhat to our alarm, as the prospect of having to go in search of pus left behind in the recesses of a deep axilla was formidable. The explanation, however, was that the abscess cavity, having permeable lung above and below, was speedily obliterated.

The other case I saw with my old pupil and friend, Dr. Liston, of Tewkesbury, and I will let him tell the story of the case in his own words.

C. T., schoolboy, aet. 10.

First seen, December 21, 1902, with rigors and high temperature. Well-marked signs of croupous pneumonia in left lung on 22nd.

History of influenza at school commencing a fortnight before and apparent recovery.

The pneumonia invaded the left base first and gradually spread to the apex; there was a crisis on the ninth day. Temperature remained normal for one day only, and then assumed a suppuration type. The physical signs now began to vary in a capricious manner from day to day as regards auscultation, but there was a steady displacement of the heart to the right.

Three attempts at aspiration were made without result, one in the left eighth interspace in the anterior axillary line, one in the mid-axilla, and one just below the inferior angle of the scapula.

The first attempt was made on December 26, on which day there was absolute dullness of the whole left base up to the angle of the scapula, complete absence of vocal fremitus and marked oegophony. The puncture was made with a medium-sized trocar of Potain's aspirator just below the angle of scapula.

As the very marked displacement of the heart continued, with great dyspnoea and suppuration chart, two further punctures were made on January 10, both in the eighth interspace, one in anterior axillary line, the other in mid-axilla. Nothing was drawn off. On the 11th a marked area of resonance was noticed in the left base, especially in front, and the left apex was absolutely dull, with no breath sounds or vocal fremitus or resonance down to the fourth rib.

Sir W. Broadbent saw the patient on the 13th.

Under his direction an exploratory puncture was made high up, the trocar entering the thorax in the anterior axillary line just below the lower border of the pectoralis major, with the arm held out at right angles to the body. Pus was immediately struck, and about one pint there and then drawn off; an incision was made in the site of the puncture, and a short large tube inserted. The chart remained one of suppuration until the 26th, when the tube was removed, the sinus dilated with sinus forceps, and a huge collection of sloughs removed; a tube of larger calibre was then inserted.

The temperature now remained normal until the night of February 11, when the tube was out and could not be returned, and the temperature was 102·4. An anaesthetic was administered

the opening again enlarged, and tube re-inserted. The temperature came down to normal and remained so.

On March 11 the tube was discontinued as the discharge was clear glairy fluid, and patient's recovery was uneventful. He was out on April 14.

His physical signs are now (July) practically normal, his entry of air being good all over, and his heart area normal in extent and position.

The fluctuations in the auscultatory physical signs mentioned by Dr. Liston, when carefully gone into, had a sufficient resemblance to those described in the first case to warrant the conclusion that there was fluid in the fissure exercising pressure upwards and downwards. Finally, the amount was so large that both upper and lower lobes were consolidated by compression.

SOME POINTS IN THE CLINICAL HISTORY OF EFFUSION INTO THE PLEURAL CAVITY

Medical Society's Proceedings, 1881

THE signs indicative of effusion into the pleural cavity are well known and easy of recognition, but a brief enumeration of them is necessary for the purpose of this communication.

Physical signs.—When one side of the chest is full of fluid and the lung is compressed into a small compact mass lying against the mediastinum, there is absolute dullness on percussion, with entire absence of respiratory murmur, diminished vocal resonance, and extinction of vocal vibration. There is usually tubular breathing between the scapula (about the level of its spinous process) and the spinal column, which is gradually lost as the ear or stethoscope recedes from this part, though it is often distinctly audible under the clavicle. Round the base of the lung, from front to back, there is usually silence. There will be more or less evidence of displacement of the mediastinum, heart, and diaphragm. The affected side of the chest may bulge a little, and give increased comparative measurement, but this is not so significant as the diminished movement in respiration. Such movement as may be observed will be upwards rather than expansile.

In estimating the significance of these deviations from the physical signs afforded by healthy lung, it is necessary to bear in mind that they are not simply the effects of the interposition of so much fluid between the lung and the chest-wall, but also of the effacement of the lung as a sound-yielding organ. There can be no respiratory murmur when the lobules and bronchiae are so compressed that no air enters them, and there can be no to-and-fro movement of air in the larger tubes to give rise to tubular or bronchial breathing; these tubes, again, are open for a very short distance, and cannot, therefore, conduct to the chest-wall consonant vibrations, respiratory or vocal, transmitted along the bronchial ramifications from the larynx or trachea.

Dullness.—If we have the opportunity of watching the gradual accumulation of fluid in the pleural cavity the necessity of taking into consideration the lung as well as the fluid becomes still more evident.

The line of dullness, though it changes with the position of the patient, is not level either in the erect or in the recumbent position, as we find it, for example, in hydro-pneumothorax. The lung does not only float, it also shrinks towards the root, the effect of which is most evident in the most distant part, that is, the base and the convex surface lying against the lateral aspect of the chest. When, therefore, the chest is partially full of fluid the line of its upper limit is curved. In the erect position it rises in a parabolic form on the axillary aspect; in the recumbent posture the line is concave upwards and forwards, and the dullness, especially on the right side, extends further forwards along the base than the fluid could well rise, the explanation being that the thin edge of the lung is withdrawn from between the arch of the diaphragm and the chest-wall.

As the fluid rises the vocal resonance and vibration are usually exaggerated over that part of the chest-wall with which the lung is still in contact, and when the chest is nearly full, so that the only resonant area in the recumbent position is a small patch below the clavicle, the resonance here is curiously tympanitic in character.

Breath sounds.—Sometimes when the pleural cavity is apparently quite full of fluid, the dullness on percussion being absolute and general and the vocal vibration extinguished, respiratory murmur can be heard on the posterior aspect of this side of the chest, most distinctly at a short distance from the spine, but conducted round towards the axillary surface. This may possibly be due to adhesions, but this is extremely rare, and far more commonly the respiratory murmur heard is produced in the other lung and transmitted through the tense mediastinum to the fluid, and by it conducted to the chest-wall. In children it is the rule that the respiratory sounds, especially rhonchi of the lung in action, are heard all over the side of the chest which is filled with fluid. I mention this because I have known an eminent physician direct the puncture to be made at a disadvantageous part, in order to avoid adhesions presumed to exist an account of respiratory murmur. If there are adhesions, either they will bind the lung so closely to the chest-wall that resonance will persist, or this not being the case, the lung will be so far compressed that air does not enter; it cannot therefore produce vesicular breath sounds. The only evidence upon which respiratory sounds of vesicular or lobular origin audible over the side of a chest full of fluid could be admitted to be produced in the compressed lung, would be the presence of elements, such as fine crepitation, absent over the sound lung.

In the account just given of the physical signs indicative of effusion into the pleural cavity it has been carefully stated that

they are present when the lung recedes, first by its own elasticity and later under the pressure of the advancing fluid, until it is compressed against the posterior mediastinum. But the lung does not always so recede; sometimes it is held by adhesions, more frequently it is prevented from shrinking by consolidation more or less complete, or by congestion, and then, instead of being compressed into a small compass around its root, the lung is immersed more or less deeply in the fluid. We should expect this to modify the physical signs, and this is the case.

For many years I have noted the fact that, in some cases of effusion into the pleural cavity, instead of silence or at the most feeble and distant tubular breathing below the scapula posteriorly and round the lateral aspect of the chest to the front, as is the rule, *loud bronchial breathing* is heard all over the affected side of the chest. I have had from time to time the opportunity of seeing post-mortem examinations in which this had been observed, but without reaching a satisfactory explanation of the variations from the more usual absence of respiratory sounds. My ideas, however, on the significance of persistent general loud bronchial breathing in the presence of extensive pleural effusion were cleared up only by M. Potain, from whom I learnt that it is indicative of imperfect collapse of the lung.

The chief point, then, to which I desire to call attention is that, while the ordinary signs of effusion into the pleural cavity, dullness on percussion, extinction of vocal fremitus, diminution of vocal resonance, and limitation of bronchial breathing to the region of the root of the lung, show that the lung retreats and shrinks before the fluid, loud tubular breath sounds at the base of the lung posteriorly and over the lateral and anterior aspect of the chest show that the lung has not entirely retreated, but that it retains a certain volume and is more or less deeply immersed in the fluid. It will be seen at once that the conditions are exactly such as will explain the conduction of bronchial breathing and of the voice to the surface of the chest. The lung, not having collapsed, is nowhere very far removed from the chest-wall, the bronchial tubes, moreover, remain patent, and the lung substance around them has lost its spongy texture and is solidified—all circumstances which favour transmission outwards of sonorous vibrations. These conditions have other consequences; it is when they are present that aegophony is heard most distinctly and over the largest area; the thin layer of fluid between the lung and chest-wall, which, by intercepting some vibrations and transmitting others, gives rise to this modification of the vocal resonance, and which can only exist temporarily at an early stage of the affection when the

lung shrinks pari passu with the rise of the effusion, may persist long when the lung is large and consolidated. In some of these cases there may even be a degree of vocal vibration at a period when the amount of fluid effused is sufficient to give dullness on percussion over the entire lung.

Paracentesis.—This question of the condition of the lung in effusion into the pleural cavity has a direct practical interest. A moment's reflection shows that when the lung is large and consolidated and deeply immersed in the fluid paracentesis is likely to be of comparatively little service, and this for two reasons; there is in the first place comparatively little fluid to remove, and in the next the removal of the fluid leaves untouched what is probably the primary and more important condition, the consolidation of the lung. A third reason for not performing this operation is that experience has shown that these are the cases which often speedily get well without it. The first statement that there is no great amount of fluid to be removed in the circumstances described was illustrated by a case recently under my care in St. Mary's Hospital, in which this condition existed, and in which nevertheless it was found necessary to tap the chest on account of urgent and increasing dyspnoea; only 30 oz. of bloody fluid could be obtained by the aspirator from a very large-chested man, although the suction power employed was greater than I consider to be advisable.

For some time I have been led by these considerations to put off paracentesis in these cases as long as possible, and I have found that they belong to one of two classes, the serious and dangerous on the one hand, the very favourable on the other. The effusions into the pleural cavity, common in kidney disease, are often accompanied by congestion and partial consolidation of the lung, which prevent it from collapsing; so again are effusions which rapidly become purulent, and this is especially the case in the early stage of acute empyema in children. Setting aside these cases, however, which are easily recognized, the signs indicative of a large congested lung deeply immersed in the fluid are prognostic of rapid absorption. I have now seen this in a sufficient number of instances to enable me to predict with considerable confidence the recovery of the patient without paracentesis and in a comparatively short time.

In watching these cases I have usually observed that one of the first steps towards recovery has been a rather sudden disappearance of the tubular breathing, and the substitution of the more ordinary signs of simple effusion, so that more than once on proposing to demonstrate the condition to students I have found it gone. As there is no return of resonance at any part it is probable that the

congested lung has relieved itself by diffusion of serum into the pleural cavity, and that the amount of fluid here is actually increased.

The fact that the presence or absence of loud and distinct bronchial breathing over all parts of a pleural cavity full of fluid and especially over those parts most remote from the root of the lung, has an important bearing on the prognosis of the result, and on the employment or not of paracentesis, removes this sign from the category of useless diagnostic refinements, and, indeed, I do not admit that any diagnostic refinements are useless. The habit of realizing as minutely as possible the condition of diseased organs, which is a statement in other words of what is meant by nicety of diagnosis, cannot fail to be of service. I will only add that the significance of the sign referred to only becomes definite when the dullness on percussion extends over the entire half of the chest; tubular breath sounds are heard over a great part of the affected side when the pleural cavity is only partially full of fluid.

Rules for Paracentesis.—It may be well, in conclusion, to state briefly the rules which, speaking generally, guide me in recommending the performance of paracentesis, although they do not differ from those usually laid down.

[1] In the absence of urgent symptoms I do not interfere until the chest is full of fluid—i.e. till the dullness is universal over the affected side, with evident displacement of the mediastinum; and when this condition has been reached, or when it is found to exist, I usually wait a few days, in some cases ten days or a fortnight, unless the fluid is still on the increase, in order to afford an opportunity for absorption, I delay longer when, from the presence of tubular sounds over the entire half of the chest, I conclude that the lung is consolidated; because, as I have explained, there is less fluid to be removed and because it is usually quickly absorbed.

When there are urgent symptoms, such as dyspnoea, either constant, or induced by very slight exertion, or coming on in paroxysms without assignable cause, there should be no delay in withdrawing the fluid. We should not wait, even if the pleural cavity is not full. Sometimes the fluid is limited by adhesions, and the upper part of the chest on the affected side may be resonant, while at the same time the heart and the mediastinum are displaced, and the heart is greatly impeded by pressure. Or there may be disease of the heart, and the fluid, though not in such quantity as to be of itself a source of danger, or even of inconvenience, may add fatally to the heart's difficulties. In kidney disease, again, it may be necessary—or, at any rate, very useful—to tap the chest before it is full. Dyspnoea constitutes an emergency which overrides

[1] *Lancet*, 1884, I.

nearly all other considerations. Bronchitis or congestion of the opposite lung, again, must be held to constitute an urgent symptom, calling for the removal of the fluid earlier than might otherwise have been deemed necessary, especially when there is violent cough and blood-streaked expectoration. I recommend tapping early when the patient is phthisical, unless it is the affected lung which is compressed by fluid. There is some reason to believe that compression for a length of time checks, if it does not arrest, disease in the lung; and I may remind you, although it is not a parallel case, that in acute tuberculosis if one lung is compressed by fluid it escapes. I recommend early tapping, again, in old people. On the other hand in children it is better to wait for a short time; if the case is not one of empyema, the fluid will probably be absorbed.

The spot to be selected for the puncture is the eighth space in a line with the angle of the scapula. We should never try to remove the whole of the fluid; this is impossible and unnecessary, and the attempt to effect it would do harm. A greater proportion can usually be withdrawn without distress when the effusion is comparatively recent than when the chest has been full for some time. In the latter case frequent partial emptyings are to be preferred. In the case of a young man, aged 26, in whom the left half of the chest was known to have contained fluid for six months, 48 oz. were withdrawn by aspiration at the first tapping, three days later 24 oz., ten days after this 18 oz., the patient recovering fair expansion of the lung. As a contrast to this I may mention the case of a man, aged 76, who was admitted into the hospital on September 24, 1875, aspirated to the amount of five pints on October 6, and discharged well on October 17.

CLINICAL LECTURE ON A CASE OF HYDATIDS OF THE RIGHT LUNG SIMULATING PNEUMOTHORAX.

Lancet, 1878. Vol. II

Gentlemen,—A clinical lecture is most useful when the subject is a case such as you are likely to meet with from time to time in your every-day work. Cases such as that I have to bring before you to-day are rare, and you may never see another like it; but some of you have followed it with so much interest that I am sure it will not be unprofitable to put into form the lessons we have together learnt from it. I should have preferred taking, first, two cases of effusion into the pleural cavity which we have watched, one of which has been treated by aspiration, the other without; so as to have taken you from a simple and common form of disease to one which is rarely met with, and whose symptoms and signs constitute a highly complex problem. Our patient, however, is dead; and it is better to study the case while it is fresh in our recollection, and we have not to depend entirely on notes, which, full and accurate as they are in Mr. Tucker's record, cannot, some weeks hence, give us the vivid picture now present to our minds of the poor suffering girl who has been the object of so much real anxiety to those of you under whose immediate care she was.

First of all, let us look at the lungs and liver, which are here for inspection; they are the only parts with which we need concern ourselves.

The liver is rather large, weighing 4 lb. 6 oz. The upper surface of the right lobe was firmly adherent to the diaphragm at its posterior part, and a piece of the diaphragm, roughly circular in shape and about two inches in diameter, remains attached to the liver, having been cut away to avoid tearing the organ. This marks the situation of a subjacent hydatid cyst, of about the size of an orange. There is nothing remarkable about it, and the only particular I need mention is that it contained only two or three small secondary cysts.

The right lung was firmly adherent to the walls of the pleural cavity at every part. The pleura over a great part of the surface was thickened and red, but the surface which had rested on the

diaphragm was thickly coated with curdy, yellow pus, which had formed a localized empyema here about half an inch deep, and exudation of the same character extended upwards on the costal aspect for a short distance, averaging about an inch from the base of the lung. These particulars are less conspicuous now that the parts have been kept for a few days, but the substance of the lung is seen to present an enormous cavity, large enough to contain a foetal head or a pair of fists, and in this the thick, white gelatinous wall of a hydatid cyst, which had originally excavated and filled the cavity, but which was found after death lying collapsed within it, together with muco-purulent matter, and broken-down cheesy material. Its internal surface presents a number of nodules, and it contained a few loose daughter cysts. Water injected into the bronchial tube entered the cavity freely by numerous small apertures.

The situation and relation of the cavity are of considerable interest. Above it there is a portion of the apex of the lung, about an inch in thickness, condensed a little, but containing air. At a part which we may suppose to have been about the level of the fourth rib, all lung-structure had disappeared except near the root, and the walls of the cavity all round the thoracic parietes consist of little more than thickened pleura. Down the mediastinal aspect behind the root, was a cushion of lung little damaged, and at the base there was a certain amount of lung-substance, but this was consolidated by long-standing pressure and recent inflammation. The cyst-wall touched the diaphragm over an area of about two square inches near the posterior border.

The other lung was normal in structure, but the bronchial tubes were filled with frothy mucus, and the lining membrane was deeply stained. A small hydatid membrane also was found impacted in the bronchus, carried thither no doubt by an inspiratory current of air.

We shall see how these pathological conditions explain the symptoms observed during life.

The patient, a girl aged nineteen (I am now making use of Mr. Tucker's excellent notes) was admitted into the hospital on September 19. The family history was good; her personal history free from attacks of serious disease, and from evidences of weakly or damaged constitution. Two months previously, however, she had pain in the chest and right side between the shoulders; she had also for six weeks been suffering from diarrhoea, and had been in the country for a fortnight on account of it, but her strength did not appear to have been greatly affected. Indeed, judging from her appearance, she must have been the picture of health up to

the time of the present illness. Her face and limbs were round and full, and her colour was good. Nine days before her admission —i.e. September 10—she felt something give way in her chest or epigastrium while she was carrying a child, and had just run upstairs with it in her arms. She thinks she was insensible for two or three minutes. She had to take to her bed at once, and had been confined there ever since, except for an hour or two occasionally. During these nine days she had severe pain in the chest, vomited frequently, was unable to keep food in the stomach, and had diarrhoea; she suffered also from severe headache, sleeplessness, and feverishness.

On her admission, the above, with the exception of the vomiting, were the symptoms complained of, and she was seen to be suffering from dyspnoea. The temperature was 102·5° in the evening, 101·8° next morning; pulse 120; respiration 38.

When I first saw her on the 20th I was at once struck by the aspect of distress and by the frequent breathing; and on the chest and abdomen being exposed it was at once seen that the breathing was exclusively thoracic; the diaphragmatic movements were restricted, so that the abdomen was almost passive during respiration, while the expansion of the thorax was excessive. That this was not due to peritonitis was evident from the absence of tenderness and the supple and natural feel of the abdomen generally. The right hypochondrium, however, and the epigastrium were indicated by the patient as the seats of severe pain, and there was great tenderness on pressure here and across the abdomen at this level to the left hypochondrium, so that the size of the liver and spleen could not be estimated by palpation. The right side of the chest was in pain, and its movement scarcely so free as that of the left. Over the left lung the resonance was normal and the respiratory murmur greatly exaggerated, suggesting at once the idea that this lung was compensating for deficient respiration elsewhere and doing the work of both. On the right side, over the front of the chest, the percussion-note was peculiar, and the respiratory murmur was wanting; posteriorly the resonance was of a lower pitch above the spine of the scapula than on the left side, while below this level there was dullness. There was little entry of air over the dull area, and at the lower part scanty coarse crepitation was heard. The vocal resonance was exaggerated.

We may interrupt the relation of the case for a moment to say how far we were able to carry the diagnosis at this period. We set aside definitely enteric fever, which had been mentioned when the patient was brought to the hospital, on account, no doubt, of the diarrhoea and abdominal tenderness, though spots also had been found, and the tongue was suggestive of this disease. We

excluded also pneumonia: the acute symptoms had lasted too long; there was too much pain, and it left unexplained the thoracic respiration and many of the physical signs. Diaphragmatic pleurisy, again, though it was obviously present, causing the breathing to be thoracic, was inadequate to account for the whole of the phenomena. Pneumothorax, with consecutive pleurisy, suggested itself, but the resonance, though peculiar, was not tympanitic, and a tap on the chest-wall while the stethoscope was applied at another point did not give the characteristic ring. When the back of the chest was examined the dullness below the scapula, and especially the presence of distinct though scanty crepitation at the base of the lung posteriorly seemed to exclude pneumothorax, and prevented further tests from being applied, as the crepitation showed that the lung was in contact with the chest-wall at a part from which it would, unless held down by adhesions, be far removed were the pleural cavity full of air. All we could say then was that the disease was in the right lung, that it was acutely inflammatory, and that the inflammation involved the diaphragm and perhaps the peritoneum at the upper part of the abdomen. A grave prognosis was given.

For the next two days the patient had less pain and felt better. The pulse on the 23rd had come down to 108, and the respiration to 29; the temperature, however, was high, especially at night, 103° F. and 104·5°; in the morning falling to 100·4° and 100·8.° It was found that she could lie more comfortably on the right side than on the left, but that she had most pain when lying on her back. It is the rule in acute painful inflammation in one half of the chest that the patient can lie more comfortably on the affected side than on the other, as the weight of the body restrains the movements which give rise to pain, and free play is left to the healthy lung. Usually, however, the recumbent position is also comfortable, and the sufferer lies alternately on the back and on the affected side. In this respect, therefore, there was something exceptional.

On the evening of the 23rd she was very much worse; the pain in the right side was most acute; the pulse rate was 160; the respiration 60 per minute, and short and catching.

I was called to her on the evening of the 24th, having been out of town in the meantime, and found the anxiety of my resident only too fully justified. More severe suffering could scarcely be witnessed, and the extremely frequent and weak pulse and the rapid catching breathing indicated imminent danger. An examination made now revealed nothing new in the abdomen, which, as before, took little or no part in the respiratory movements, so that

respiration was altogether thoracic. The left lung was doing supplementary work, and its respiratory murmur was greatly exaggerated. Over the front of the right lung the resonance was excessive, without being markedly tympanitic, down as far as the fifth rib, where there was an abrupt transition to absolute dullness ; nothing definite was made out by auscultation ; there was certainly no respiratory murmur, but no distinct abnormal sounds were present, while the loud breath sounds from the other lung were heard. The apex of the heart was displaced to the left of the mamma, where it could be felt beating violently. When the patient was raised into the sitting posture, the level of dullness on the anterior aspect was found to rise about an inch. A minute examination was not justifiable in the patient's condition, and after ascertaining that there was dullness and silence over the lower part of the right thorax posteriorly, attention was directed simply to the application of a test for pneumothorax, the presence of which had been suggested by the signs already enumerated. The ear or stethoscope was applied to the back of the chest about the mid-scapular level, and at a corresponding point anteriorly a coin was pressed against the thoracic wall and slightly struck by the edge of another. The sound rang through the chest, and it was almost as if the ear were against a gallon jar which had received a smart tap. The peculiar ring was recognized by the least practised ear, and the difference on the two sides was unmistakable. Here apparently was an explanation of the whole of the phenomena. When the sudden pain and sensation as if something had given way were felt, after hurriedly carrying a child upstairs, rupture of the lung had occurred, probably at some point previously diseased, and air had escaped into the pleural cavity. On this there had been pleurisy with formation of pus, and the condition with which we had to deal was pyo-pneumothorax. This was our inference. Now that we know that the condition was quite different, we can see that we neglected certain indications—the extensive dullness over the back of the right chest, the crepitation at the base of the lung. The history, however, was exactly that of pneumothorax going on to the complication of purulent effusion, and the physical signs were corroborative of the conclusion that this was the condition present.

When air obtains entrance into the pleural cavity by rupture of the lung or otherwise, the lung collapses ; and we have a kind of drum—a large air containing cavity, with walls easily thrown into vibration, yielding on percussion and auscultation signs such as we might expect. There is excessive, or rather unnatural, resonance, sometimes, but by no means always, tympanitic ; occasionally, indeed, the percussion note is of a character which might by an inexperienced

observer be named dull. I may remind you also that we may have tympanitic resonance over the chest without pneumothorax. When air passes into and out of the cavity with every breath there will be amphoric respiration, which sounds like blowing into a large bottle or jar. This is not common, however; and the chief sign is that we relied upon—the ringing echo which is heard when the ear is applied to one part of the chest and a smart tap is given to some other part, the most characteristic effect being obtained when a couple of coins are used in the manner described. Frequently the action of the heart is heard across the pleural cavity, accompanied by a similar ring. When with air there is fluid, which in these cases is usually pus, splashing as of water in the gallon jar, to which we have so often referred, may sometimes be heard when the patient is quickly shaken. This "succussion" phenomenon we failed to elicit in our case.

Having made the diagnosis of pyo-pneumothorax, the next question was what should be done. Clearly the first thing needed was that the pain should be relieved, and half a grain of morphia was at once injected under the skin. But as it seemed possible that there might be great tension of the air in the pleural cavity from the opening in the lung being valvular, and allowing air to enter during inspiration, while it prevented its escape on expiration, and that this tension was the cause of the excessive pain, an aspirating needle was ordered to be introduced, and air or fluid to be drawn off. The morphia gave great relief, and the patient had a good night, but the attempt at aspiration only resulted in the abstraction of a few drops of sanguineous fluid, which, under the microscope, showed blood-corpuscles and large exudation-cells.

Next day she was better, and she continued to be much easier from the 25th to the morning of the 30th, this being entirely due to the suppression of pain by the hypodermic administration of morphia; and the pulse became less frequent, and the respiration less hurried and painful, while the temperature, on the contrary, continued to range high, reaching 103° on most evenings, on one 104.° Free, but not profuse, expectoration of muco-purulent matter set in rather suddenly on the 26th, and continued; it was taken to be pus from the pleural cavity making its way out by the opening which had admitted the air. The obvious presence of mucus was remarked, but the pus of an empyema escaping by a bronchial tube acquires much mucus in its passage outwards, poured out no doubt by the irritated bronchial and tracheal mucous membrane, so that unless the amount of pus is very large, it becomes more or less glairy. The same thing was recently noted in the pus of a vertebral abscess, which made its way into a bronchus in the

case of a little boy under my care in the Albert ward. No hydatid membranes were seen in the sputum; they could scarcely have escaped observation had they been present in any quantity, as the expectorated matter was carefully scrutinized at every visit. During these five days we several times considered the question whether we should treat the case as we should have done one of simple empyema, by making a free opening into the chest through one of the lower intercostal spaces, and trying to drain and cleanse the pleural cavity. My experience of this practice in pyo-pneumothorax has been unfavourable, and it was rejected, especially as the pus was escaping, and the heart and mediastinum were not displaced so far to the left as to indicate pressure, but only to the extent to which they would be carried by the elasticity of the left lung on air being admitted into the right pleural cavity, as has been beautifully demonstrated by Dr. Douglas Powell.

A few observations worthy of record were made during this period. The remarkable amphoric ring was several times demonstrated, but we could not obtain splashing sounds by gently shaking the patient. One day sibilant cooing rales were heard over the front of the left lung, but more distinctly over a small area at the apex of the right. This we supposed to be due to adhesions which had prevented the collapse of this part of the lung. Had not the sounds been louder in the diseased side they would have been considered to be conducted from the sound lung, as was a loud rhonchus produced in the left, but heard also over the right side of the chest.

On the 30th the patient became rapidly worse, and died. We have already seen the results of the post-mortem examination. It remains only in a few words to show how the condition we found simulated so closely the signs of pneumothorax. This, indeed, will be plain to all of you.

The hydatid parent cyst was burst by the effort of carrying the child. As its contained liquid escaped—whether rapidly or slowly we do not know, for the evacuation must have taken place before the patient came under our observation, but probably it was during the vomiting—as the hydatid fluid escaped, the cyst would collapse in folds to the lowest part of the cavity in which it was lodged, where it would lie together with the inflammatory products exuded. The cavity itself could not fall in, since it was held open by adhesions all round, and as the hydatid fluid escaped its place would be occupied by air entering through the patent bronchial tubes we saw at the post-mortem examination. Although, then, we had not the pleural cavity itself containing air and pus, we had a cavity with similar contents, smaller, it is true, and having an

additional thickness of wall, but still of enormous size, and capable of giving similar physical signs.

We may, in conclusion, ask two questions. Was a diagnosis possible? and could we have done more for the patient had an accurate diagnosis been made? I will answer the latter question first. Had I known exactly the state of things I should certainly have ordered a free opening to be made into the cavity, and I am sure that we should thus have given the poor girl a better chance of recovery. In reply to the first I would say that a diagnosis, though difficult, was, no doubt, possible. I say so because I think that perhaps you or I might make out a similar case were such a one to come before us. In order, however, to interpret the complex signs and symptoms offered by disease, we must be prepared by personal experience or by acquaintance with the recorded experience of others, and until this case came under my care I did not possess the knowledge that hydatid disease could so nearly simulate pneumothorax.

AN ADDRESS ON DILATATION OF THE STOMACH

Delivered before the Harveian Society

British Medical Journal, 1893, Vol. II

Dyspepsia gives rise to a great variety of symptoms, and is responsible for much discomfort and suffering and even misery, if we take into account the depression of spirits with which it is frequently accompanied. I propose to deal here with one of the consequences of dyspepsia, which, at the same time, aggravates its injurious effects, and constitutes a serious obstacle to remedial measures. This is dilatation of the stomach, which has, moreover, symptoms of its own and requires special treatment.

There are various degrees of gastric dilatation, and I should almost be prepared to say different kinds. The increased capacity of the organ, the imperfect collapse and contraction when digestion is completed, and the exaggerated area of resonance before or after meals are common to all cases, but in some patients splashing is easily elicited, and when the tube is introduced, at whatever interval after food, a quantity of ill-smelling liquid will be withdrawn; in others a splash is heard only for two or three hours after food, and after this time the stomach will be found quite empty by the tube when the line of resonance is as high as the fifth space. It is not easy to draw a line between mere distension and dilatation, theoretically we should say that actual dilatation existed when the stomach tolerates passively the presence of gas, and does not clear itself of contents after digestion.

Causes.

The causes of dilatation of the stomach are such as give rise to indigestion and flatulence, and first among them stands improper and injudicious food and feeding.

Improper and injudicious food and feeding.—Over-feeding generally is a common cause of dyspepsia, which may go on to the production of dilatation. Appetite, which would be satisfied by a reasonable amount of one or two kinds of food, is reawakened by a succession of dishes: the stomach is thus completely overloaded, and is incompetent to deal with the mass of material it

receives. Bulky, farinaceous articles of diet, certain green vegetables of the cabbage tribe, which give rise to flatulence, with copious draughts of liquid, will distend the viscus, and, by frequent repetition, impair its contractile power. The largest stomach I ever saw post-mortem was in the body of an Irishwoman, whose staple diet was potatoes and tea ; it extended from the left hypochondrium to the right iliac fossa. Habitual taking of food between meals is a practice which is ruinous to digestion ; like other organs, the stomach requires a period of rest. Food, again, taken in a condition of exhaustion or extreme fatigue is never properly digested, and the man of business who thinks he is taking exercise, which in some degree neutralizes the ill-effects of confinement to his office, by walking home from the city, not infrequently adds to them by fatigue which impairs his digestion. The rush from work to food, and from food hastily eaten back to work, is, again, a frequent cause of indigestion, as is also the hurry from breakfast to catch a train to town.

Functional Derangements of Nervous Origin.—But the stomach is astonishingly tolerant of excessive and improper food, and adjusts itself to irregularities of meals and untimely feeding with extraordinary facility, and, on the other hand, the severer forms of dyspepsia and gastric dilatation are met with when the meals are wholesome in character, moderate in amount, and regular in time. Some other cause, therefore, than errors in diet must play a very important part in their production. Hereditary or congenital tendency to dyspepsia, injury to the stomach by improper food in infancy and childhood, will account for some cases, but a more potent influence is disturbance by the nervous system, taking effect in some cases on the secretion of the digestive fluids, in others on the muscular contractions of the stomach. A large proportion of the dyspeptics who consult us, and of those who suffer from dilatation of the stomach, are neurotics, and the neurosis is the cause of the stomach trouble, and the nervous symptoms are not the consequence of indigestion, as is so commonly assumed.

Anatomical Conformation.—In dilatation of the stomach, however, my observation has led me to conclude that there is very frequently another factor, an anatomical disposition, which hinders the expulsion of the products of digestion. This is when the pylorus is suspended high up in the epigastrium by a short lesser omentum, which also limits its movements. Under such circumstances if the stomach is distended and overloaded it is dragged down by the weight of its contents and an acute flexure is formed at the junction of the duodenum and pylorus, which constitutes an obstacle to the passage of the chyme. I have many times seen, when peristalsis

has been provoked by handling, an enormously dilated stomach define itself through the thin abdominal walls, making a sharp bend at about two-thirds or three-quarters of its length from the cardiac end, the pyloric portion passing directly upwards or even upwards to the left, while the contraction travels from left to right, and on arriving at the pylorus evidently encounters obstruction which it is unable to overcome. Mr. Mayo Robson has recently pointed out another mechanical cause of dilatation of the stomach in adhesions which have formed as a result of ulcer or of perihepatitis.

Symptoms.

The symptoms to which dilatation of the stomach gives rise are extremely varied. They include all those attributable to indigestion, but it would only lead to confusion to enumerate them, and I shall specify only such as are more or less characteristic of dilatation, and they may be roughly classified as gastric, mechanical, reflex, and as due to ptomaine poisoning.

It is not always easy to say to which of these classes particular symptoms ought to be referred, and they no doubt overlap, but it will facilitate description and discussion to attempt some kind of classification.

Of the gastric symptoms proper the most characteristic is *copious vomiting*. One meal after another is taken through the day, or, in some cases, for two or three days, with more or less discomfort, but without sickness, and then apparently the whole is returned, the quantity sometimes being enormous. The vomited matters are usually of a brown colour and offensive odour, with a floating scum. This, of course, is a symptom commonly present in malignant or other stricture of the pylorus, and the diagnosis between dilatation secondary to disease of the pylorus and simple dilatation is often difficult. The complete absence of free hydrochloric acid from the vomited matters, or from the contents of the stomach when withdrawn by a tube, has been relied upon as evidence of cancer; but even if this point were absolutely trustworthy, which I doubt, it is very difficult of establishment. The presence or absence of sarcinae, again, is not conclusive either way; there is always an abundance of fermentative organisms, and sarcinae may or may not be among them. Time and the results of treatment will ultimately establish the diagnosis, and in most cases the judgment must be held in suspense for a while.

In other cases the vomiting is frequent and occurs at varying intervals, after each meal or every night, the stomach, however, never completely emptying itself, so that whenever food is taken it is received into a quantity of fermenting material, and itself

undergoes fermentation instead of digestion. Eructation of offensive gases is sometimes a symptom, but in bad cases the stomach has lost the power of expelling gases. The breath may or may not be offensive. The state of the tongue does not afford much assistance in the diagnosis. The most common surface appearance which it presents is a thin, pale, moist coat, which might be described as slimy or greasy looking, while it is large, soft, flabby, and indented at the edges. Almost every variety of fur may, however be met with, and in some cases the tongue remains quite clean. Usually the appetite is impaired, and not uncommonly there is an absolute disgust for food, so that the proper nourishment of the patient becomes a matter of extreme difficulty. On the other hand, there may be a spurious appetite, the patient having a sensation of hunger and sinking very soon after food, and sometimes an imperious craving for food at all times of the day and night.

Effects on the Heart.—One of the most serious of the mechanical effects of dilatation of the stomach is upward displacement of the diaphragm and pressure upon the thoracic viscera. The heart is often very greatly embarrassed by pressure and displacement, and when the organ is fatty or dilated, or weak and flabby, dilatation of the stomach may determine a fatal arrest of its action. In one case at St. Mary's Hospital, a patient admitted for extreme breathlessness and weak and frequent action of the heart, and found on examination to have considerable dilatation of the stomach, died apparently from the effect of this upon a sound heart, no other cause of death being discovered post-mortem. I had ordered the stomach to be washed out, but collapse supervened before this could be done. A hearty or indigestible meal may give rise to sudden death, or the dilatation may render fatal exertion or emotion which would otherwise have been survived.

Palpitation of the heart after meals is a common accompaniment of a dilated stomach. More frequently the heart's action is irregular, sometimes violent, at others feeble; occasionally there is intermission. As a rule the patient is acutely conscious of irregular or intermittent action of the heart of gastric origin, whereas when it is present from cardiac disease, he knows nothing about it. Probably reflex disturbance may play a greater part in the production of palpitation, intermission, and irregular action of the heart than pressure, since cardiac disturbance may be very severe when the enlargement of the stomach takes a downward direction, and the line of resonance is not very high.

A symptom more distinctly traceable to pressure upon the heart and lungs is oppression and difficulty of breathing on lying down

at night. In the recumbent position the weight of the liver and the abdominal viscera generally is more or less thrown against the diaphragm instead of falling away from it, and both heart and lungs are additionally embarrassed by the increase of pressure upon them. Sleeplessness is a very common effect.

Sleeplessness.—A still more common form of sleeplessness is produced by a minor degree of gastric dilatation. The patient sleeps on going to bed, but at 2, 3, or 4 a.m. is awakened, and remains awake for some hours, perhaps till morning. The explanation is that the stomach does not completely expel its contents, and in the course of the night fermentation takes place with evolution of gas, which, by aggravating the pressure on the diaphragm, disturbs and prevents sleep. The subject usually does not recognize flatulence, or discomfort arising from flatulence, as the disturbing influence, but anything which causes the eructation of gas removes the inability to sleep. It may here be remarked that flatulent distension of the stomach produces its worst effects when the patient ceases to be conscious of the flatulence as such. The discomforts arising from the presence of gas are usually due to the efforts of the stomach to get rid of it, and it is when the stomach suspends these efforts and allows itself to be passively distended that the pressure and reflex effects give rise to serious trouble.

Nocturnal Asthma.—The mention of the form of sleeplessness just considered leads up to the consideration of nocturnal asthma, which is the most characteristic of the reflex symptoms. This comes on at about the same time in the night, and almost certainly from the same cause—fermentation, during the first hours of sleep, of imperfectly digested food, with the formation of gaseous and irritating products. The spasm of the bronchial tubes, however, cannot be due to pressure, and must be a result of a reflex from the gastric branches of the pneumogastric to the motor fibres of the bronchiae. The excitement or aggravation of nocturnal asthmatic paroxysms in predisposed individuals by indigestion or flatulence, late or heavy meals, or certain articles of diet, is matter of frequent observation; when the attacks are habitual, dilatation of the stomach may be suspected as the cause. It is usually in adults at or after middle age that asthma is set up by dilatation of the stomach. There may be no bronchial catarrh whatever, or the gastric affection may precipitate the occurrence of asthma as a complication of bronchitis. The attacks are sometimes very severe. Occasionally nocturnal spasm of the larynx may be provoked in the adult by dilatation of the stomach, and may appear to threaten life.

Vertigo.—Vertigo, sometimes so severe that the patient has to cling to railings in the street, or to help himself by the chairs and tables in

crossing a room, is not uncommon as a result of dilatation of the stomach. It may or may not lead up to a paroxysm of vomiting. When premonitory of an attack of sickness, the giddiness may be attended with nausea, faintness, pallor, and cold perspiration, which is sometimes so protracted as to threaten life before relief is obtained by an attempt, often futile, to empty the stomach. In other cases, with an extreme sense of giddiness, there is no faintness whatever, and the patient can carry on a conversation or continue writing when he could not move from one chair to another without staggering. The dependence of the vertigo on the state of the stomach is shown by the fact that eructation of gas brings instant relief, and the patient who is not troubled with nerves learns to wait for this with equanimity. This vertigo *a stomacho laeso* is distinguished from auditory vertigo by the fact that there is no apparent translation of external objects. The patient is conscious that his head swims, but the room does not turn round, nor the floor tilt up, nor the bed topple over. The attack, moreover, does not come on with overwhelming suddenness, and I do not remember a patient actually falling.

Nightmare, violent starting of the limbs on going to sleep, are other illustrations of reflex disturbance.

The symptoms which may be set down to ptomaines absorbed from the fermenting contents of the stomach are headache, depression of spirits, and morbid ideas. Many of the discomforts attributed to "biliousness" or "liver derangement" are really due to this and other affections of the stomach. Altogether the sum of misery to the patient and his family and friends occasioned by dyspepsia is a very considerable item in the unhappiness of civilized society.

Other effects of dilatation of the stomach which must not be forgotten are loss of flesh and a dark sallow hue of the complexion. The loss of flesh is often such as to be suggestive of malignant disease, especially where there is obstruction of the pylorus, and the dark discoloration of the face might easily be taken as indicative of cancerous cachexia. It can scarcely be called pigmentation, as it clears up too rapidly when the gastric affection is relieved. The conjunctivae may or may not be stained.

Diagnosis.

The definite diagnosis of dilatation of the stomach rests ultimately upon physical signs. These are an abnormal extension of the area of gastric resonance with a tympanitic echo of the heart sounds over this area, the more or less ready production of splashing, and the tinkling of water falling into the distended viscus when th

patient is made to drink, and, finally, the actual measurement of its capacity by means of the stomach tube.

Sometimes the stomach can be seen to bulge out at the epigastric region, forming a rounded prominence across the upper part of an abdomen not otherwise much distended, or, indeed, excavated. The lower limit can be recognized by inspection, and can be defined accurately by stroking the abdomen downward with the flat of the hand, when a difference of resistance is felt as the fingers glide off the distended viscus. This stroking method of palpation often affords valuable corroborative information when no bulging is visible.

Percussion.—In percussing out the stomach it is well to examine the patient in two positions—sitting up and lying down. In a few obscure cases additional information may be obtained by turning him also first on one side, then on the other. In the upright position the weight of its contents may drag the stomach down and away from the abdominal wall, so that the tympanitic note is indistinct, and its extent, and especially its upper margin, indefinite; while in the horizontal position the fluid gravitates to the back, which becomes the lowest part of the viscus, and the whole anterior surface is in close contact with the wall of the abdomen, when the characteristic resonance is easily elicited and is found to encroach upon the chest. Account must always be taken of the period after meals at which the examination is made, and of the amount and character of the food last taken, in forming an opinion as to the degree of dilatation present and in making comparison between one day and another.

Almost the only question to be solved when there is undue resonance in the epigastrium and left hypochondrium is how much of it is due to stomach and how much to colon. Even when the line of resonance is as high as the fifth space it may be that the stomach is carried up by the colon or by gaseous distension of the small intestine, or atony of the diaphragm may invite displacement upwards.

The first point in differentiating stomach from colon resonance is the uniformity of the percussion note over a given area from above downwards and right and left, beginning in the epigastrium, or just below the heart, where it is probably gastric. The tap must be light and glancing. A perpendicular stroke, especially if at all forcible, brings out a note belonging to the general abdominal resonance, or to any dilated viscus in the neighbourhood, and effectually confuses gastric and colon distension. But percussion alone is never to be trusted absolutely. It is checked in the first instance by placing the stethoscope at some point within the resonant area, and giving sharp flips with the nail over its entire extent. So long

as exactly the same ringing note reaches the ear from the various points at which the tap is made, it is probable that they are all upon the same viscus. The stethoscope will be shifted, and the click of two coins, one placed on the skin and struck by the other, may be substituted for the flip with the finger nail. In all these trials it must be borne in mind that the note belongs to the underlying viscus, whatever it may be, at the point where the tap is made. If, for example, the stethoscope is placed in the epigastrium, and the tap is made over a dilated transverse colon lower down, the colon note will be heard, enfeebled perhaps, but not altered in pitch. Let now the stethoscope and point of impact change places, and a totally different note will probably be heard.

Another check is afforded by *the conduction of the heart sounds.* When the stomach is dilated to such an extent as to displace the diaphragm upwards, the heart and stomach will be in close contact, with only the diaphragm intervening. The heart sounds are then conducted, or rather echoed, with a tympanitic ring all over the stomach, thus helping to define its limits. It is worthy of note that the second sound is always much more distinct and ringing than the first.

The splashing sound may be elicited in various ways. In the upright position the ear or stethoscope is placed in contact with the anterior abdominal wall at or near the epigastrium, and a sharp motion is then communicated to the body. The splash of the liquid in the dilated stomach is usually very distinct. When, however, the stomach is full, or nearly full, of liquid, and there is no room for gas, splashing cannot be produced, but the naked ear may feel a powerful wave of fluid impinging against the abdominal wall. Care must be taken not to mistake borborygmi, easily induced in the colon, for splashing.

In the recumbent position a sharp push with the fingers from behind over the false ribs will make the liquid splash, or the patient may be partially rolled over. When all the corroborative evidence attainable is desired in a difficult case, the patient may be made to drink while the observer listens at the epigastrium and in its vicinity. If the stomach is dilated and is not too full of fluid, the gurgling and tinkling produced as the water enters it and drops into the liquid which it already contains will often give a good idea of the size of the organ.

In some cases *peristaltic action of the stomach* can be provoked by handling, and the dilated viscus can be felt to become firm and prominent under the hand. This, indeed, is not infrequently also visible, and the contraction can be seen to travel slowly from left to right up to the pylorus. It only occurs, of course, when there

is actual obstruction at the pylorus from thickening or from an acute flexure as already described. No peristalsis can be elicited when the dilatation has resulted from atony of the muscular coats of the stomach or from passive distension.

Colonic peristalsis travels from right to left. It is very rarely that a diagnosis can turn on this, but in one case of a patient sent to me by a distinguished physician as dilatation of the stomach due probably to malignant disease of the pylorus, a contraction proceeding from right to left at once indicated obstruction and dilatation of the colon, which was successfully treated.

The final demonstration of the existence of dilatation of the stomach is effected by the employment of the stomach tube. The contents are first withdrawn, and warm water rendered alkaline by carbonate of soda is then poured in pint by pint till the stomach will hold no more. The normal capacity ascertained by this method varies, the average being about one or two pints. Dilatation exists when the amount which can be introduced is three pints or over. In severe cases six or eight pints have been poured in.

Treatment: Dietetic.

In treatment of dilatation of the stomach induced by a single excessive and indigestible meal or by a comparatively short course of overfeeding, or by an alcoholic debauch, it may be sufficient to fast absolutely for twenty-four or forty-eight hours to effect a cure, and in all cases in which the cause has been overfeeding or improper food, or food taken at a wrong time, an extremely strict and meagre diet for a few days will be the best starting-point for treatment. No advantage would, however, result from fasting or low diet in cases of neurotic origin. Patients of this class will, it is true, often experience extraordinary relief for a time when first released from the obligation of taking food, but the effects are disastrous later. The inadequate supply of food leads to weakness, and with weakness comes impairment of the digestion and aggravation of the nervous susceptibility and irritability. A return of pain after meals is attributed to some article of diet, which is thereupon expunged from the list of permissible foods, and the patient enters upon a downward course of starvation, weakness, emaciation, indigestion, and pain, which often ends in an acute illness of some kind.

Having said so much with regard to the dieting of patients whose dyspepsia and gastric dilatation are primarily neurotic, the subject may be continued. The object we set before ourselves must be, as I have said elsewhere, not to level down the diet to the digestive capabilities of the stomach but to level up the digestion till it can

deal efficiently with the amount of food required for the due support of the nervous system. No hard and fast rule can be laid down. A careful study of the patient's idiosyncrasies will be required, and the diet must be adjusted to these. Speaking generally, such a patient will digest better food which he relishes, even if it have the reputation of being indigestible, than the most digestible and scientifically prepared food which he eats by order and dislikes. A very common experience is that he is tempted by a good dinner, eats largely and indiscriminately, and then, instead of a bad night and great discomfort which he thinks he has deserved, he sleeps well and feels all the better for his indiscretion. A very important point will be to disabuse the patient's mind of the idea that pain after meals necessarily indicates that the food has been unsuitable; this will be more difficult to effect with women than with men. One day and under one set of circumstances anything will agree, on another day and under different circumstances nothing is digested. The general directions to be given will be to restrict the amount of fluid taken at meals, to eat starchy food in very moderate quantity, as it is bulky and lends itself readily to fermentation, to take only one vegetable at a meal, not to eat when exhausted or especially when excited or anxious, or to eat very sparingly and simply at such times, not to jump up from meals and rush off to work of any kind. Subject to such limitations as the above the food should be varied so as to tempt the appetite, and the resources of good cooking may be freely employed for the same purpose.

With regard to *stimulants*, a small amount as part of the two principal meals is usually helpful to the digestion, by giving a fillip to the stomach, which may or may not increase the secretion of gastric juice, but more certainly augments the energy of the churning movements of the stomach. Experience is the best, and, indeed, the only sure guide in the choice of a stimulant. Good spirit, well diluted, is simpler than wine or beer, and can neither start fermentation nor supply fermentable material. For myself, I have never been able to recognize the superiority of whisky over brandy; and I am disposed to think that the reason why the former has been so largely ordered by medical men is that it is nastier, and perhaps, therefore, less likely to be taken in excess. Good whisky is, no doubt, better than bad brandy, and vice versa. When any good, sound, genuine wine agrees with the patient, it may be taken in preference to spirit.

When the dilatation of the stomach has been primarily due to what may be called gastric causes, excessive and improper food and the like, the rules with regard to diet must be applied more strictly, and, as already stated, temporary starvation and a very limited

amount of food for some time may be most useful. The rules will be: regularity in the hours of meals, and strict avoidance of intermediate food; little fluid with meals, and very little starchy matter, the object being, of course, to avoid distension of the stomach, whether by the bulk of food and drink introduced, or by the generation of gases. A familiar out-patient formula is "no beer, tea, or potatoes." Only one vegetable should be taken at a time. The application of rules may usually be left to the common sense of the patient, but when this mental quality is entirely lacking it may be necessary to write down the dietary. Stimulants are less necessary than in the case of neurotic patients, and may often be forbidden altogether with great advantage.

An expedient often of great service in relieving some of the effects of dilatation of the stomach, and sometimes contributing to a cure, which may perhaps best be mentioned in connexion with diet, is *drinking hot water*. This has become a common practice, and hot water is taken with meals or after or before or between meals for the relief of indigestion, the reduction of obesity, and various other purposes. The special object for which I have employed hot water has been the prevention of sleeplessness and nocturnal asthma. As already explained, flatulent distension is a frequent cause of inability to sleep on lying down, and especially of waking up at a given hour. In the latter case, remains of the last meal not carried on into the duodenum ferment and evolve gases. A large tumbler of very hot water sipped at bedtime stimulates the stomach to contract; almost always a certain amount of gas is expelled at once, and frequently sufficient to allow of sleep in cases where flatulence has prevented it. The contraction has the further effect of carrying any contents of the stomach forward into the small intestine, so that the copious draught of hot water washes out the stomach and prevents the lingering behind of contents which would undergo fermentation. Sometimes the addition of carbonate of soda, with perhaps sulpho-carbolate of soda, will add to the efficacy of the hot water, or a draught containing these salts with ammonia, compound tincture of chloroform, and other carminatives may be given half an hour before the hot water. Numerous cases of habitual and aggravated sleeplessness, and several of nocturnal asthma, have been effectually and permanently relieved by simple measures of this kind. Sometimes, however, the stomach is incapable of responding to the stimulus of hot water, even when aided by carminatives, and then the discomfort and distress are aggravated.

While speaking of the employment of hot water, it may be remarked that taken on an empty stomach in the morning the effect

is entirely different; it is rapidly absorbed, and, passing through the tissues, acts as an eliminant. For stout and overfed people this is often very beneficial, but weak, thin, neurotic subjects are weakened and ultimately depressed by it.

Treatment by drugs.—Several objects have to be held in view in the treatment by drugs of dilatation of the stomach. The two principal are to rectify the chemical processes taking place in the stomach, so as to promote digestion and substitute peptonization, which is not attended with evolution of gases, for fermentation, which gives rise to the formation of acids of various kinds and the setting free of gases, and to promote the contractile energy of the muscular walls, so that not only may the churning movements be efficiently performed, but the contents completely expelled at the end of digestion, and passive distension be resisted.

When there is pain after food it may be necessary to give bismuth and magnesia or soda, with perhaps a small dose of opium before meals, and when there is heartburn to give carbonate of soda or other alkalies; when, again, there is flatulent distension, carminatives may be required. But it should be recognized that measures of this kind are only palliative, and that in employing them we are not treating the disease, but only some of its consequences. It is, of course, useful, and indeed necessary, to procure the eructation of gases which are distending the organ, but, if no other result is sought, the benefit is fugitive, and the remedies in time lose their efficacy. So, again, with alkalies; unless the acid fermentation is stopped they are required in increasing quantity, and the amount taken may come to be enormous—if I rightly understood a professional friend the other day he was taking six drachms of bicarbonate of potash per diem. A good method for the administration of antacids is in the form of lozenges, which are to be slowly sucked, so that alkaline saliva is carried down in quantity as well as the drug, as was pointed out by Sir William Roberts.

Pepsin, again, and especially peptonized foods, which often find a useful place, are also to be looked upon as chiefly palliative.

The two main objects in the treatment of dilatation of the stomach, the prevention of gaseous distension and improvement of the contractile energy of the muscular coats, are attained more or less perfectly by many familiar mixtures and pills, and different members of the Harveian Society will have been led to place confidence in one or other combination of antiseptics and tonics. I will simply indicate some which have appeared to me to be of service.

When there is eructation of gas having the odour of sulphuretted hydrogen the best remedy is, according to my experience, sulphite

of soda. The sulphurous acid disengaged probably combines with the sulphuretted hydrogen, and also kills the particular microphytes which disengage this gas. At any rate the foul smelling eructations have always promptly ceased. The sodium sulphite may be given in doses of 5 to 10 grains, with carbonate of soda and nux vomica between meals.

Another combination which I have frequently employed with conspicuous benefit has been sulphocarbolate of soda in doses of 5 to 10 grains with carbonate of soda, spirits of ammonia, and gentian. It may be given in the early stages of a case when there is distension and discomfort at a certain interval after meals, with eructations. Ginger, chloroform, or peppermint may be added to the mixture to promote the expulsion of gases. If there is gastric or intestinal catarrh, or when the evacuations are pale, phosphate of soda in half-drachm or drachm doses is a useful addition; or 1 or 2 drachms of phosphate of soda may be given early in the morning in hot water with taraxacum.

Other useful remedies are creasote and carbolic acid, which are best given in pill form with strychnine. Advantage may also be taken of the bactericidal powers of mercury to arrest fermentation and the formation of ptomaines. A very useful combination is gr. $\frac{1}{24}$ each of hydrarg: perchloride, or biniodide, and strychnine with ♏j or ♏ij of creasote or carbolic acid in a pill which may be given before or between or after meals. When arsenic is indicated as a tonic by the condition of the nervous system gr. $\frac{1}{24}$ to gr. $\frac{1}{18}$ of arsenious acid may be included.

In a large proportion of patients, careful diet on the lines indicated and persevering treatment will bring about improvement of the digestion and removal of the dilatation of the stomach; but relapses are frequent and difficult to prevent, and cases are met with in which, from mechanical causes or from entire loss of tone and contractility in the muscular walls, no impression is made on the condition. Fortunately a resource still remains open to us in the stomach tube.

Washing out the Stomach.

The process of washing out the stomach is a very simple one. The fluid usually employed is a weak solution of bicarbonate of soda, about a drachm to the pint of lukewarm water; 10 to 20 grains of sulphocarbolate of soda may be added when the contents are offensive in character. The introduction of the tube is at first disagreeable, and provokes retching and perhaps vomiting, but the patient very soon learns to pass it for himself. He should sit bolt upright in a chair, bending the head slightly forward, and the tube

should be carried boldly to the lower end of the pharynx, the patient being told to make swallowing movements, and in the intervals to breathe deeply. When the extremity has reached the stomach, sufficient liquid should be poured into the funnel to fill the tube, without which it will not act as a siphon. The tube is then pinched, and the funnel end is lowered so as to be well below the level of the stomach, when the water introduced and the contents of the viscus will flow out. The liquid should now be poured in till a sense of discomfort and fullness is produced (the amount required being noted), upon which it is made to return by again lowering the extremity of the tube. When the patient has become accustomed to the proceeding, the flushing may be repeated once or twice till the gastric contents have been completely cleared out, and any mucus clinging to the walls has been washed away.

Care should be taken that the whole of the fluid introduced is withdrawn, or discomfort and sharp purging may follow. The capacity of the stomach may thus be definitely ascertained, and the amount of liquid it will hold should be carefully noted. It will usually be found to diminish, at first rapidly, later more gradually.

The relief afforded by washing out the stomach is usually very striking. When irregular and intermittent action of the heart has been one of the most troublesome symptoms, this is at once suspended, so that for a time the pulse becomes quite regular, for how long I have had no opportunity of noting, probably till after the next meal, and the heart's action becomes steadier and stronger from day to day, and the patient can take exercise with greater comfort.

Sleeplessness is usually much relieved. I have not yet found it necessary to order the washing out at bedtime, but it would probably be still more efficacious in preventing disturbance of sleep if done then.

Vomiting is of course put an end to; there is no further occasion for it. Flatulent distension also is usually got rid of more easily by eructation. But more important than the removal of these symptoms is the return of appetite and the disappearance of the loathing of food which is so common.

By way of conclusion, two or three cases may be related in outline.

Case 1.—A lady, aged about 45, whom I saw in consultation with Mr. J. T. Mould. The special feature in her case was vomiting. This usually occurred in the evening or during the night, sometimes more frequently, and was very copious. She had reached an extreme degree of emaciation and weakness; her skin hung in folds and wrinkles, and her complexion had a deep yellowish brown

tint. She might have sat for a picture of cancerous cachexia. She was very energetic, and insisted on going out, but could only crawl for a few hundred yards. The abdomen was deeply excavated, and the enormously dilated stomach could be seen in its upper segment. The lower border descended from the left hypochondrium to near the umbilicus, where the pyloric third bent suddenly upwards towards the liver. On handling, the muscular walls contracted and the stomach defined itself through the thin abdominal wall, while a peristaltic movement travelled slowly from the cardiac to the pyloric end. On succussion a heavy fluid wave could be felt to impinge on the hand or stethoscope when the stomach was full; when it contained only a small amount of fluid a loud splash was heard. The suspicion of cancer of the pylorus was heightened by the presence of a small firm tumour just where this end of the stomach dipped under the liver. It appeared, however, to be too mobile and to move independently of the pylorus, and it was probably the gall bladder containing a calculus or calculi. Mr. Thomas Smith saw the patient two or three times, and the question of operation was carefully discussed. Some relief, however, had been afforded by washing out the stomach, and it was decided to give this treatment a thorough trial. It was undertaken by Dr. Callender, who had done the preliminary washing out. The patient very soon learnt to pass the tube for herself, and the result was a complete recovery. The last I heard of her was that she mingled with the crowd at the Royal wedding.

Case 2.—In June I was asked to meet Mr. Bryant, the President of the Royal College of Surgeons, Dr. Crisp, and Mr. Lyster at the Bolingbroke Pay Hospital to discuss the question of operation upon a young man, aged 27, who had been suffering for two years from vomiting. He was much reduced in flesh, weighing only seven stone (his height being 5 feet 8 inches). There was tenderness on pressure in the epigastrium, and in this region was a firm, solid mass, not moving freely in respiration, and not very well defined as to its lower border. An important consideration was whether or not this was a malignant growth. At the age of the patient, however, cancer in this situation would not have taken two years to bring about a fatal termination, and it was thought to be more probably a mass of inflammatory exudation consequent upon a gastric ulcer which had nearly perforated. Adhesions and exudation round the pylorus would obstruct peristalsis, and prevent the stomach from emptying itself, and retention of the gastric contents would interfere with the healing of an ulcer. It was decided, therefore, to wash out the stomach, which was carried out by Mr. Lyster. The vomiting ceased, and the patient left the

hospital a stone heavier, and has since been able to return to partial work in the city.

In dilatation of the stomach of neurotic origin, or in which the principal symptoms are cardiac, while the results of washing out the organ are not so palpable, they are often very striking.

In one case a gentleman, aged about 60, had apparently nearly lost his life from embarrassment of the heart, brought on by hurrying to catch a train after a hasty meal: he had been brought home pulseless, cold, and cyanosed. For some time afterwards he was liable to attacks of faintness and breathlessness in the night or after food, and the action of the heart faltered after the least exertion. Great dilatation of the stomach was found to exist, with considerable upward displacement of the heart. This had, no doubt, been kept up by incessant feeding, which an anxious wife had insisted upon against the advice of the medical man, but when the diet was properly ordered the dilatation persisted sufficiently to give rise to serious discomfort from heart symptoms. Here a few washings out by Dr. Callender were followed by remarkable relief, although the gastric contents were on the first occasion which I witnessed only a few shreds of undigested food and a small quantity of mucus.

The moral of this communication is the desirability and usefulness of a careful physical examination even in an affection which is so generally regarded as a purely functional derangement as is dyspepsia.

THE CAVENDISH LECTURE ON SOME POINTS IN THE TREATMENT OF TYPHOID FEVER

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SOME explanation is perhaps necessary of the choice of this subject. Surely, it may be said, the treatment of typhoid fever is sufficiently well understood. It has been carefully laid down by great clinical teachers, and, while new suggestions are multiplied on every hand, it may be objected either that they relate to details which afford no basis for a revision of principles, or that, on the other hand, they are of so radical, and even revolutionary, a character that time is required before a judgment can be given with regard to them. But there is no finality in medicine, no conclusion in therapeutics, in which we can for any length of time rest and be thankful, and no one who has written upon typhoid fever would claim to have said the last word upon the questions involved in its treatment. For my own part, I should desire to be understood not as speaking with authority, but simply as offering a contribution to the subject, the result of personal observation and thought. A special reason for a revision of our ideas as to the treatment of typhoid fever arises out of the more extensive and profound knowledge obtained in recent years of the part taken by specific microbes in the production of disease and in the determination of its course and duration. The life history of these micro-organisms has been followed out, the mode in which they influence the human system has been more or less definitely ascertained, the conditions under which their virulence is intensified or attenuated have been investigated, the processes by which their action on the organism is resisted and their lethal influence brought to an end have been studied.

Again, while the bacteriologist has been engaged in identifying and studying the particular bacterium, or coccus, or protozoon which sets up a given disease, the chemist has been working for the discovery of germicides or bactericides which might destroy the morbific agents, and of substances which might protect the human system from their attacks, and naturally physi-

cians have been eagerly inquiring how this knowledge may be turned to account in combating the diseases which they have to treat, and whether there may not be found in it resources which may render to medicine some such service as the researches of Sir Joseph Lister have rendered in surgery.

The chemist, again, has placed at the service of the medical man products which possess the marvellous power of lowering the temperature of the body. These, at first sight, seem to be the very weapons we require with which to subdue fever. We have all looked with hope to these antiseptics and antipyretics, and it will be useful to compare notes on the methods we have employed and on the results we have obtained; and typhoid fever is a favourable field for such comparisons. But it is not simply on this account that I have taken it as the subject of this lecture. It is a disease possessing great clinical interest from the variety of features which it presents in different cases, and from the opportunities which it gives for careful observation and judicious modifications of treatment. Probably more frequently in it than in any other disease the medical man may turn the scale in favour of the patient, when the issue of life and death lies in the balance, by watchfulness, knowledge, and skill, and there is no better test of clinical sagacity and capacity than the conduct of a case of typhoid fever.

Feeding.—Nothing need be said as to the general management of the patient; nothing too emphatic could be said as to the importance of careful nursing. Very little, again, is necessary to be said on the subject of feeding, although, on this, success largely depends. The popular idea is that, the disease being one characterized by weakness, the remedy is nourishment and perhaps stimulants, and that the more the sufferer can be induced to take the better. One of the first tasks of the medical man is usually to prevent over-feeding. In the early stages of the disease any liquid offered is eagerly taken, especially if it is cold, and quantities of milk or milk-and-soda-water may be given altogether in excess of the powers of the digestion. Curds and other irritating products are thus formed which give rise to flatulent distension, nausea, and sometimes vomiting, and aggravate the intestinal catarrh.

An average amount of food would be about two pints of milk and one of broth or beef-tea in the twenty-four hours, to be given eight or ten ounces at a time at intervals of about two and a half or three hours by day, and rather less frequently at night, so as not to interfere too much with sleep, the discretion of the nurse being exercised in this respect. But, while naming the above amount and character of nourishment as that to be aimed at, no absolute

rule can be laid down as applicable to all cases. There are patients, for example, whose dislike for milk, with probably a corresponding incapacity for digesting it, is such that they cannot take it in any form, and we have to depend on broth and beef-tea or other meat extracts or preparations throughout. We may, again, be compelled to give the nourishment more frequently from the first, because only a small amount can be taken at a time, and as the case progresses the intervals almost always have to be shortened on this account.

The Stools.—An absolutely indispensable guide to the feeding of the patient is regular and systematic inspection of the dejecta. This is the secret of success in the treatment of typhoid fever. The medical man ought to see every stool which is passed, or, if this is for any reason impracticable, at least one motion every day. It is only by personal examination that he can know the amount and significance of the diarrhoea—whether, for example, it is copious or only frequent, each action being scanty, and can tell when and in what way to step in and interfere. The character of the motions, again, often reveals the cause of any special disturbance of the bowels. A particularly offensive smell and unusual colour may indicate septic processes in the intestinal contents, or it may be seen that some article of diet is playing the part of an irritant—as, for example, strong beef-tea. If undigested milk appears in the evacuations, however finely the curds may be divided, irritation of the intestinal tract is sure to follow, and probably diarrhoea. In this case measures should be at once taken to ensure the better digestion of the milk. Soda-water or lime-water may be added to it with a view to prevent premature coagulation, or it may be diluted with barley water, which does not permit of the formation of large solid curds. More effectual than either of these expedients is the peptonization of the milk. This unfortunately destroys its natural flavour and gives it a more or less bitter and disagreeable taste, so that patients usually dislike it, which interferes with its usefulness. When it is necessary to peptonize the milk it should be done thoroughly, according to the method described by Sir William Roberts.

Stimulants.—The subject of stimulants, again, will require only a very few words. Were I speaking only to this Society I should need to do no more than express my general agreement with the conclusions set forth in the excellent address of your President, Dr. Donald Hood. The part which stimulants take in the treatment of typhoid fever is chiefly to carry the patient through the later stages of the disease when the nervous system is exhausted, the circulation failing, and digestion all but suspended, or as an

accessory to cold bathing. Many cases do not really require stimulants from first to last; in no case should they be given at once as a matter of routine. The indications which tell that they will be of service, and that the time has come for their use, are frequency and low tension of the pulse and dryness of the tongue, together with nervous and muscular prostration and mental confusion. The amount given should be small at first—one and a half, two, or three ounces of brandy—and it should be subdivided and taken with or after the milk or other nourishment. As the asthenic symptoms become more pronounced the amount will be increased, but always in a guarded manner. My own observation has led me to look upon ten ounces in the twenty-four hours as almost the maximum from which good effects may be expected. Beyond that amount stimulants appear to make little impression on the symptoms, and it should be an object of solicitude not to reach the full quantity prematurely. A change of stimulants often does better than an increase in the quantity, such as the substitution of whisky, or port, or old Madeira, or Malmsey, or champagne for brandy. It is a common idea that drunkards and persons habitually consuming a good deal of alcohol require large amounts when suffering from typhoid fever. I am not at all sure that they benefit by it; the vascular and nervous systems are so habituated to it that they make no response. Such patients are exceedingly likely to die however freely they may be plied with stimulants, and I am disposed to believe that they would have a better chance on abstinence than on alcohol, nux vomica or strychnine being given from the first. When stimulants are really necessary the good effects are seen in a lowering of the pulse-rate, the volume and tone being improved; usually also the temperature is somewhat reduced, the nervous system is steadied, and there is more restful sleep. A noteworthy point is that there is no odour of alcohol in the breath. Whenever this is persistently present the stimulant is almost certainly doing harm instead of good. A regulated amount of brandy or other stimulant does not preclude the administration of a supplementary dose when called for by sudden faintness or failure of strength, or when the patient is exhausted by an action of the bowels.

Treatment.—While according to nursing and feeding and to the judicious employment of stimulants their full importance in the treatment of typhoid fever, the supervision and regulation of these do not exhaust the functions of the physician. He may very frequently influence favourably the course of the disease and contribute to the recovery of the patient by the use of drugs, and by other means outside the province of the nurse or beyond a mere attitude of expectancy, and he must at times, if he is to save life, interfere

energetically. In no class of cases can it be said more forcibly that we do not treat the disease but the patient, in none is routine treatment less applicable. In the course of typhoid fever numerous injurious influences are in operation, dangers arise of a very varied character and there are diverse tendencies to death. We ought to have in our minds a clear perception of the processes which in a given case underlie the symptoms. This alone will give precision to our efforts to counteract their ill effects. With this view a classification of the morbid agencies which together constitute the disease may be of service.

Sleeplessness.—Before entering upon this, however, there is one point, relating to general treatment, which may be conveniently discussed. It is, what should be done in case of sleeplessness, which is so common? While deprecating a premature resort to sedatives of any kind in typhoid fever, as in other conditions, when cold sponging fails to sooth the patient, and when it is clear that the restlessness and want of sleep are not due to flatulent distension of the stomach or other removable cause, it is, in my judgment, better to secure sleep by drugs than to allow the patient to pass wakeful and miserable nights. The drug I prefer is opium in some form or other. Phenacetin or antipyrin will often relieve the initial headache and give sleep, and phenacetin as the least mischievous member of the group may be given once or twice, but these substances are depressing, and, at the outset of a long and exhausting illness, any deduction from the sum total of the patient's forces is to be avoided. Chloral, again, distinctly lowers the vigour of the heart's action, and relaxes the arterioles, imitating thus the effects of fever on the circulatory apparatus. Experiment, moreover, has shown that it paralyses the phagocytes and renders them inert in the presence of bacilli which they would otherwise attack. Chloral is therefore inadmissible, and, it may be added, is usually ineffectual. Bromidia, which may be looked upon as chloral in disguise, often ordered I fear in ignorance of its composition, I should emphatically condemn. Bromides are less open to objection for occasional use, but are seldom of real service. When, therefore, a remedy for sleeplessness is needed it should, in my judgment, be sought in one or other of the preparations of opium or morphia. It has been objected that opiates derange the digestion. This has not been my experience, but they may be employed to disguise the effects of flatulence, the result of digestive derangements which ought to have been corrected.

General Treatment.—Returning now to the various factors which enter into the disease, we can recognize three more or less distinct causes for the symptoms, each of which is attended with dangers

of its own: (1) *the operation of the poison;* (2) *the intestinal lesions;* and (3) *the protracted high temperature.* The poison is the primary cause of the disease, and we may consider first effects traceable more or less directly to toxic influence on the system, and the means by which they may be counteracted. The typhoid bacillus primarily sets up inflammation in the gland follicles of the intestine, but its activity is not confined to these structures. The bacillus is present also in the blood, and, whether in the blood or in the glands, it secretes, or produces by disintegrating the albuminoid substances in which it lives, a ptomaine which gives rise to fever, headache, prostration, and various effects on the nervous system. The febrile temperature is probably the direct result, and the best measure of the activity of the bacteria which have entered the blood and been carried throughout the system.

The first question to be considered is whether by any form of treatment the typhoid germs can be destroyed, or their action arrested, or their toxic influence counteracted. There has never been a time when physicians have not entertained the idea of cutting short fever. As the zymotic origin of fever gained acceptance efforts in this direction took more and more the form of antiseptics; and now that the typhoid bacillus can be isolated and its life-history followed, and that the effects of different agents on its vitality and virulence can be studied, it is natural that such efforts should be renewed. In endeavouring to estimate the probabilities of their success we must bear in mind that when symptoms appear the microbe has already eluded or overcome the phagocytes, protective alexins, and other defensive agencies, and has obtained an entry into the blood and tissues, where it is actively multiplying and producing its specific ptomaine. Now, it is exceedingly improbable that any antiseptic could be introduced into the blood in sufficient proportion to destroy the bacillus, or even materially influence its activity, which would not at the same time profoundly and injuriously affect the blood corpuscles and the gland and nerve cells. We get no encouragement from bacteriological research to look forward to the destruction of the microbes when once in possession, and clinical observation still endorses the conclusion that the fever runs its course in spite of remedies of the antiseptic class.

A method of arresting the multiplication and activity of the bacilli is suggested by the mode in which the termination of the attack is effected. The cessation of a fever is not brought about by exhaustion of the pabulum on which the bacillus thrives, but by the accumulation in the blood and tissues of some product of its action upon their constituents which arrests its further activity, and apparently protects the organism from future attacks of the disease, just as

the alcohol produced by fermentation brings the growth of the yeast organism to an end. Possibly an antitoxin may be obtained which, injected under the skin, may anticipate the termination due to the formation of such a substance in the system. This, which is different from the introduction of a protective alexin producing immunity from invasion by the bacillus, has not yet been done, however, in typhoid fever, though apparently diphtheria has been in some measure brought under control by such a method.

Antiseptics.—But recognition of the fact that no antiseptic has yet been shown to be capable of destroying the bacteria in the blood does not carry the implication that antiseptics have no place in the treatment of typhoid fever. Ptomaines are not only produced in the blood, but in the intestinal canal, whence they are absorbed into the blood. And there are here other microbes at work besides the typhoid bacillus. The exudation from the inflamed Peyer's patches and from the intestinal catarrh affords a pabulum for various septic agents, and their ptomaines are added to the typhoid poison proper. Apparently some of the products of intestinal sepsis are even more deadly than the primary disease. It is one thing to follow the typhoid bacillus into the blood; it is quite another to deal with septic processes taking place in the intestinal canal, and there can be no doubt as to the benefit rendered by antiseptics employed for this purpose. A great variety of substances belonging to this class have been recommended and employed: carbolic acid, sulphocarbolates, β-naphthol, salol, chlorine, hypochloric acid, iodine, boracic acid, and borates. I have tried most if not all of them with good result, but I have come to rely almost exclusively on mercurial preparations, which I have found to be of very great service when administered for certain definite purposes and in obedience to definite indications.

Diarrhoea.—One of the effects of septic processes in the contents of the intestine is diarrhoea, the character of which may be recognized by the offensive and sometimes putrid smell of the evacuations, differing from that of the typhoid stools, strictly speaking. The colour is often dark and not unfrequently the consistence is watery with brown particles or flakes in suspension, quite unlike the pea-soup typhoid stools. The abdomen at the same time is often distended with gas, and the temperature may be considerably raised by absorption of septic products. Diarrhoea caused in this way is most frequent early in the disease and is most common in patients who have indulged in alcohol or in excess of food. It may also be due to continued consumption of solid food after the fever has set in.

The treatment I have found most efficacious in diarrhoea due

to sepsis of the intestinal contents is the administration of perchloride of mercury, which I have usually given in doses of one drachm of the solution with one grain of quinine every three or four hours for twenty-four or forty-eight hours. Calomel in repeated small doses of one-third of a grain every three or four hours is equally effectual; the diarrhoea has almost always, in my experience, been checked, the abdomen subsiding and the temperature falling one or two degrees. The entire aspect of the case is usually changed. So favourable has the effect of the perchloride been that I have often been induced to continue its administration in small doses of half a drachm three times a day through a great part of the fever. A common practice in Germany is to initiate the treatment of a case of typhoid fever by two or three grains of calomel on two successive days. Without going so far as to recommend this, I think it would prevent diarrhoea such as I have just described, and at the outset of the attack a single dose would probably be beneficial in most cases.

During the first few days of the disease, when the diagnosis is still in doubt, few patients escape an aperient of some kind; it should in my judgment be a rule of practice not to let this be of an irritant kind; a couple of grains of calomel, alone or with a grain of extract of hyoscyamus, followed if necessary by a mild saline, will clear out the bowels, carrying off both fermentescible material and fermentative organisms without aggravating the catarrh or irritating the inflamed Peyer's patches, and will at the same time disinfect the intestinal tract, while colocynth or compound rhubarb pill or liquorice powder might set up a troublesome diarrhoea.

Ptomaine poisoning. — More formidable than local irritation and aggravation of the catarrh of the small intestine are the effects on the nervous system of absorbed products of bacterial action on the intestinal contents. Other bacteria besides the typhoid bacillus may effect an entry into the blood through the lesions in Peyer's patches, but it is the ptomaines formed by their activity in the alimentary canal which constitute the poisons. Sometimes with diarrhoea of the kind just described, with high temperature and tumid abdomen early in the disease, there is either wild, maniacal delirium or great stupor and heaviness. These are cases attended with extreme danger. The good effects of the employment of the perchloride of mercury or calomel are frequently seen in the mitigation of the nervous symptoms as well as in the alleviation of the intestinal irritation. Without fever intestinal ptomaines may give rise to headache, languor, stupor, and even coma, which disappear with the clearing out and disinfection of the alimentary canal; and in the course of typhoid fever

much graver symptoms of the same kind may appear. They are entirely foreign to the normal course of the disease as usually observed, and are obviously due to some other poison than that produced by the typhoid bacillus.

I have previously related a striking example of this complication. Some years ago I was called by Dr. Ford Anderson to a young man in the second week of an illness of obscure character. A diagnosis of acute tuberculosis had been made by a physician previously consulted, but Dr. Ford Anderson considered the disease to be enteric fever, and the aspect of the patient was characteristic. The temperature, however, was low, the pulse so small and weak as to be scarcely perceptible, and the circulation in the extremities so languid that the hands and feet were greatly swollen, cold, and livid. The sounds of the heart were scarcely audible. The intellect was clear, but the weakness and prostration were such that it was a painful effort to answer any question, and the patient remonstrated against examination and could scarcely be induced to say anything except "I am so tired." There had been nothing in the course of the attack to explain the extreme depression of the nervous and cardio-vascular systems, and it could only be attributed to the action of some poison, the most probable source of which was intestinal sepsis. The abdomen was soft and supple, and there was no tenderness in the right inguinal region; the intestinal lesions, therefore, were apparently not of a serious character, and might be disregarded in the presence of the emergency. It was resolved to clear out and disinfect the intestinal canal, and three grains of calomel were given at once, and repeated next day. The asthenia was at once relieved, and the case resolved itself into a mild form of typhoid fever, which pursued uninterruptedly a favourable course.

Extreme cases such as that just related are very rare, but instances in which there is oppression of the nervous system, which is not accounted for by the temperature and is out of proportion to the severity of the symptoms generally, are not uncommon. When this conclusion is established by careful investigation it may be inferred that the symptoms are due to some poison, and I have many times seen stupor and muscular tremor remarkably relieved, and the entire aspect of the case changed, by one, or two, or three grains of calomel. There are certain rare cases of typhoid fever characterized from a very early period of the disease by frequent vomiting of small quantities of bilious fluid. The temperature ranges low, the pulse is weak, and the abdomen almost normal in appearance and feel. Such cases usually terminate fatally in the course of the second week by asthenia. Here, again, it has appeared

to me that the depressing agent was an intestinal ptomaine, and calomel has been of great service.

The intestinal lesions.—Coming now to the intestinal lesions, their severity and extent may usually be judged of by the degree of tenderness over the right iliac fossa and the distension of the abdomen, taken together with the amount and character of the diarrhoea. When diarrhoea is at all excessive, the first thing to be done is to ascertain whether it is excited by the food. Strong beef-tea in some patients appears to irritate the gastro-intestinal mucous membrane, and to hurry through the whole length of the bowel. A more common food cause is undigested milk. Diarrhoea so caused has already been considered. Another cause of diarrhoea — sepsis of intestinal contents—has also been considered. When these two sources of intestinal irritation have been eliminated it is surprising how few cases remain in which there is diarrhoea of such amount as to require control. We accept three motions a day and rather prefer to have this number unless the quantity is excessive or the patient very weak. When interference is called for, either by the frequency or the copiousness of the stools, the best remedy is a starch and opium enema; or, when the action of the bowels is frequent and irritable, but scanty, starch alone, by its soothing action, is often sufficient. I have not for years had to prescribe astringents by the mouth, and I should strongly deprecate the employment of catechu or other drugs of this class. Bismuth and opium, with or without aromatic chalk powder, or acid and opium, would be the remedies I should choose were any required. Abdominal tenderness may be relieved by a light poultice or by opiate fomentations.

In some cases there is a tendency to constipation throughout the fever. In the early stages I should not hesitate to give a small dose of calomel followed by a mild saline, which sometimes corrects the tendency, or one or two drachms of castor oil. Should the constipation persist, a small enema may be administered every second morning. These cases are often tedious and protracted and difficult to deal with, and there is a greater liability to relapse.

Tympanites.—In the course of the fever more or less distension of the abdomen very commonly supervenes. This is partly, but not altogether, an effect of the inflammation of Peyer's patches and of the intestinal mucous membrane, through paresis of the muscular coats. When accompanied by decided local tenderness and general tension it may be looked upon as due to the intestinal lesions, and it may be treated, according to associated general conditions, by cold water compresses—i.e. a single layer of thin flannel, linen, or calico kept wet with water at any temperature

thought best, or made cold by particles of ice, or, as just said, by opiate fomentations or a light poultice. But the paresis of intestinal walls which allows of general abdominal distension is not always a direct effect of the lesions in the mucous membrane. It creeps on together with delirium, stupor, muscular tremor, and subsultus tendinum, and, like them, is evidence of failure of nerve power, and the treatment required is the same. Occasionally there is acute tympanites, a quite sudden and extreme distension of the abdomen. It marks extreme nervous prostration and may be accompanied by retention of urine. When it comes on early in the disease it is one of the most fatal prognostic indications and at every period is a sign of great danger. Stimulants avail nothing against it; strychnine hypodermically may be of service, but according to my experience the remedy of greatest value is *opium*, which to be of any use must be given in considerable doses—half a drachm of laudanum, repeated in an hour or so, or even one drachm in extreme cases. I have known several lives saved in this way, and have never had reason to repent having given a large dose under such circumstances.

Haemorrhage.—One of the most important effects of the intestinal lesion is haemorrhage. The liability to this complication cannot be said to be proportionate to the pain, distension, and diarrhoea which have attended the attack, though it is more liable to occur when these evidences of intestinal disturbance have been present. Sir William Jenner pointed out with perfect accuracy that haemorrhage or perforation was to be looked for when the initial headache had been unusually severe and protracted. The ulceration in such cases, even when not particularly extensive, is liable to be deep. Enlargement of the liver is another common antecedent. When it occurs early in the attack—say, at the tenth or twelfth day—it is always attended with serious danger. After the eighteenth day, while a source of anxiety when the amount of blood lost is considerable, it may usher in convalescence, the temperature, which usually falls on the occurrence of copious bleeding, sometimes not rising again. Whenever sloughs or clots are found in the stools a careful look-out should be kept for haemorrhage. Its occurrence may often be recognized, before blood is passed from the bowel, by pallor of the face, weakness of the pulse, and a rapid drop of the temperature, and the colon may be felt to be full and heavy in the right loin.

The treatment on which I have come to rely is the placing of a large ice-bag over the right iliac fossa, the adminstration of a full dose of some liquid preparation of opium, and the subcutaneous injection of ergotin. Ten or fifteen minims of turpentine may also

be given every three or four hours. The object of the opium is to paralyse the bowel. The blood poured out into the intestinal canal excites peristalsis and the peristalsis in turn tends to prevent the formation of clot on the bleeding surface, which might seal up the vessels. It must, therefore, be arrested; and it is because opium given by the mouth appears to effect this better than morphia hypodermically that this method of administration is preferred. Half a drachm of laudanum, or its equivalent of liquor opii, may be given at once. Opium, again, seems to have a certain sustaining power. Astringents, supposed to act directly on the bleeding vessels, are useless; long before they have traversed the twelve or sixteen feet of bowel to reach the ulcers they will have combined chemically with its contents, and will have expended their power of coagulating blood or astringing tissues. When the medical man lives at a distance from the patient the nurses should be provided with an ice-bag, laudanum, and hypodermic pellets of ergotin, and have instructions to employ them immediately on the occurrence of serious haemorrhage. Great caution must be exercised in administering stimulants. The half-fainting condition affords the best opportunity for the bleeding to cease, and the longer the patient can be kept in this state with safety the greater is the chance of the arrest being final.

Perforation of the bowel is almost always fatal, but I have seen at least two cases of recovery in which, from the symptoms at the time and the formation of a dense mass of thickening between the umbilicus and the right iliac fossa, I had no doubt of its occurrence. The best chance for the patient is surgical intervention, and, if that were impossible, the administration of a large dose of opium or morphia for the double purpose of lessening the terrible shock which attends perforation and of arresting intestinal peristalsis, and so minimising the extravasation of the contents and affording an opportunity for adhesions. I should not hesitate to inject half a grain of morphia under the skin, and give thirty or forty minims of laudanum by the mouth.

High Temperature.—We come now to the third great source of danger in typhoid fever: the protracted high temperature. I have already said that I have been led to look upon the proper temperature of the typhoid fever, independent of complications, as due to the ptomaine generated by the bacilli in the blood, and this poison shares with the temperature in the production of the effects on the nervous and muscular systems. Aggravation may be caused by intestinal sepsis and by the absorption of products of inflammation from the Peyer's patches, etc. The high temperature having been long regarded as a deadly influence, it was natural

that the antipyretic class of substances should be hailed as bringing us exactly what we wanted in the treatment of typhoid fever. According to my experience they have not only failed us in this respect, but have done positive harm. They have undoubtedly the effect of relieving the initial headache, but this has appeared to me to be a very delusive benefit. One of Sir William Gull's pregnant sayings was that the removal of symptoms is not the same thing as the relief of disease. The antipyretics not only knock down the temperature, but the patient also—sometimes fatally—and I have often in cases admitted into hospital diagnosed not only the disease but the remedy. My conclusions are based entirely upon observation, but it has interested me to learn that Roque and Weill have found out that antipyrin arrests the elimination of toxines by the urine without preventing their formation. Quinine has done good in large doses as against high temperature, but its benefits have often been dearly purchased at the expense of vomiting, noises in the ears, deafness, and confusion.

The most efficacious means of controlling the heat of fever, however, is the application of water to the surface of the body. *Tepid or cold sponging* is part of the recognized duty of the nurse, and is practised night and morning even in the mildest cases. Where the temperature ranges high the sponging should be done more frequently, and it is well to time it so as to anticipate the rise which usually takes place at a certain period of the day, which will be pointed out by the chart. In such cases it will be useful to supplement the sponging by a continuous wet compress over the abdomen, frequently renewed or moistened, or kept cold by fragments of ice distributed over it. While, however, sponging, the wet compress, the ice-bag applied to the head, and similar measures are useful and grateful, they fail to exercise the desired control over the temperature when the fever is at all severe.

A resource of greater power is the *bath*, and it must be said that wherever this has been systematically tried the mortality of typhoid fever has been considerably reduced. The diminution in the death-rate was particularly conspicuous in the hands of Brand, who systematised the bath treatment. It fell from 25 per cent. to 9 per cent. Professor Tripier, of Lyons, has had equally favourable results, and the latest statistics which have come under my notice, those of Professor Osler in the Johns Hopkins Hospital, are entirely confirmatory. Dr. Cayley, again, has borne emphatic testimony to the value of bathing, and my own experience while physician to the London Fever Hospital, and in the occasional employment of the bath in consultation practice since that time, has convinced me that it saves life. The

good results of the bath treatment have long been known to the profession in this country, but it has not come at all into general use. The principal reason, no doubt, is that the cold bathing is usually very distressing to the patient, that the labour involved is great, and that in the general hospitals it is difficult to make the special provision required and to supply the additional number of nurses. There has not, moreover, been the same contrast between the results with and without bathing in this country as abroad, because our mortality was not so high.

Brand's system is to place the patient in water at 70° or 65° for twenty minutes every three hours so long as the temperature in the rectum rises in the intervals to 102·5°, cold water being at the same time applied to the head. The patient is dried immediately or only after an interval, according to the effect of the bath on the temperature and according to the strength of the patient, and he is very lightly covered. On the Continent, as I have understood, the patient gets into and out of the bath himself, and no harm seems to come of this; here and in America he is lifted carefully in and out, which adds enormously to the trouble, and at the London Fever Hospital the process was often rendered less disagreeable to the patient by cooling down the bath gradually from 90° or 85° to 70° or 65°.

I will not occupy your time by describing in detail the immediate effects of the bath; the general result is a mortality of 7 per cent. as compared with an average of 15 or 20 per cent. My own conclusion was that the disease ran a somewhat protracted course and that relapses were more frequent. Professor Osler's experience appears to have been similar as regards relapses. I will not detain you by discussing the explanations given of the good results of the bath treatment, but it is an interesting, and to me a new fact that cold bathing is found to promote the elimination of toxines by the urine. Only one observation I would make, which is that the bath acts not only by abstracting heat, but by the impression made on the nervous system, and in hyperpyrexia the shock of sudden immersion in ice-cold water or of violent affusion with ice water is the more important action of the two, and is, indeed, essential to the effect.

The continuous bath.—The latest development of the bath treatment of typhoid fever, destined, as it seems to me, to supersede all other, is the continuous bath which has been employed for several years with remarkable success by Dr. Barr of Liverpool. I have watched his records with great interest, and was so greatly impressed by the results that before appearing here I determined to see for myself the methods by which they were obtained. I paid a visit,

therefore, to the Northern Hospital at Liverpool, where I was shown cases under treatment and had the opportunity of examining the apparatus employed. This consists of what Dr. Barr calls a tank—a magnified bath with perpendicular sides raised on short legs. The size is important, not only as allowing the patient plenty of room, but because a considerable volume of water is the more easily maintained at an equable temperature. The usual temperature is 95°, but it may be lower when the fever is high and does not yield, 90° being the lowest which Dr. Barr has found necessary in ordinary circumstances. In hyperpyrexia the bath may be emptied, and cold or iced water dashed or run over the patient. The patient reclines on an Ilkley couch framework, over which is stretched a loosely-woven material which easily allows of the passage of the water through its meshes. In the newest form of the apparatus the patient can be raised on his couch completely out of the water by means of a pulley arrangement. A thermometer floats on the water, by a glance at which the nurse sees what its temperature is, and when necessary she adds hot or cold water, as may be required. At first this was done by buckets, and any excess was removed, or the tank was emptied by means of a siphon; but now the hot and cold water are supplied by pipes, and a large exit pipe four inches in diameter passes from the middle of the floor of the tank immediately under the nates of the patient. The shoulders are supported by an air cushion, upon which, again, rests another small, ring-shaped air cushion for the head, and the body is covered by a thin blanket which is brought over the chest up to the neck, as it is difficult to keep the upper part of the chest under water, and the blanket keeps it warm and moist; over the tank is a waterproof sheet.

The patient is not necessarily taken out of the tank when the bowels act or the bladder is emptied. Advantage is taken of his removal when the tank is cleansed, once in the twenty-four hours, to obtain an action of the bowels if possible, but at other times the evacuations are passed into the water. The provision made for minimizing the contamination is that under the nates there is a circular aperture in the webbing on which the patient rests, and immediately below is the large exit pipe by means of which the water receiving the stools can be run off at once before they are diffused in the bath; a considerable depth of water under the patient is consequently necessary. It would be easy to adapt to the margin of the aperture a flexible funnel communicating with the exit pipe, which would still further diminish the liability to contamination. The three patients I saw under tank treatment appeared to be perfectly comfortable; the skin of the hands and

feet was white and corrugated, as is seen in washerwomen, but elsewhere the integument had a natural appearance. The tongue invariably becomes moist within twenty-four hours, and any abdominal distension present is said to subside and delirium and tremor to quickly yield.

Dr. Barr has furnished me with complete statistics of the typhoid fever treated by him. These would be out of place here. The general result is that out of 180 cases, 43 of which had the continuous bath, 7 patients died, not quite 4 per cent., and 2 deaths could not fairly be attributed to the fever.

There is no reason to suppose that the typhoid fever of Liverpool is of a less severe type than elsewhere, and the number of cases is sufficient to establish a *prima facie* conclusion as to the results; and if there is a method of treatment by which the mortality of typhoid fever can be reduced to 2 or 3 per cent. the only question which remains for us is whether it is generally applicable. In my judgment there is no doubt as to this. I have had some experience in the ordinary bath treatment, and it appears to me that the trouble involved in the continuous bathing is much less. I hope, therefore, that a tank-room will soon be provided at every hospital, and I see no reason why a portable tank should not be constructed, provided with means of renewing and removing the water, and with all other requisite appliances, which might be kept at the different nursing establishments, to be sent out to private houses with nurses trained in their use. It should be added that Dr. Barr reserves the continuous bath for the more severe cases, but the principle of continuous abstraction of heat by water is carried out in all. The abdomen is covered by a wet compress, which is frequently moistened, and only a single thin blanket is allowed as bed-clothing. The extremities, however, may have extra covering, and may be kept warm by hot bottles, if necessary.

Résumé of Treatment.—Having analysed, so to speak, typhoid fever into the factors which enter into its symptomatology, and considered the methods by which the different processes making for the injury of the patient and tending to his destruction may be counteracted, I may conclude by bringing them together again, as they are likely to arise in the course of a case, giving briefly the treatment. The patient will, of course, be put on liquid nourishment, the amount and kind of which and the time of its administration will be clearly indicated. There will probably be a few days in which the diagnosis will be uncertain, during which it may seem that an aperient is called for. Let this take the form of one or two grains of calomel, followed by a mild saline such as phosphate or sulphate of soda. The resulting motion and every other stool

which the patient passes should be carefully inspected. Should there be severe headache, not relieved by cold to the head, ten or fifteen grains of phenacetin may be given at night once or twice perhaps. As soon as the diagnosis is formed, cold or tepid sponging will be ordered, or, rather, seeing the excellent results of this practice in the hands of Dr. Barr, a wet apron would be applied to the abdomen and kept moist instead of reserving it for special indications, the bed covering being extremely light. In no case should an eiderdown or other impervious coverlet be permitted. If the maximum temperature in the twenty-four hours does not exceed 102° or 102·5° F. no other treatment will be needed; but even then unremitting vigilance must be exercised to the last day of the disease, a report of food, drink, sleep, evacuations, temperature, etc., being submitted by the nurse at each visit.

When due care is exercised it is not likely that diarrhoea will be induced by undigested milk or improper food. Should it occur and be attributed to intestinal sepsis, liquor hydrargyri perchloridi with quinine, or fractional doses of calomel would be given, and, this failing to check the undue frequency of the action of the bowels, a starch-and-opium enema would be administered after each loose motion. With a higher temperature cold wet applications would be employed more assiduously and more extensively, and liquor hydrargyri perchloridi (half a drachm to one drachm), or one-third of a grain of calomel might be given with one or two grains of quinine, especially if the temperature rose suddenly or pursued an erratic course suggestive of intestinal sepsis, and this whether there were septic diarrhoea or not, the effects, of course, being carefully watched. Supposing the temperature to defy ordinary measures and to remain at or near 105° in spite of the wet sheet, ice compresses over the abdomen, the ice cap, etc., affusions of ice-cold water would be necessary or a large enema of iced water might be administered. This is on the understanding that the tank treatment is not available, and that the difficulties attending the employment of the cold bath prevent recourse to it. When Dr. Barr's tanks can be had, the continuous bath will be preferred in all cases in which the temperature reaches 103°.

Sleeplessness would be alleviated by ten or fifteen minims of liquor morphinae bimeconatis. As the disease advanced, if the pulse became frequent, soft, and short, brandy would be given, beginning with two to four drachms every three or four hours, the amount would be increased if the tongue were dry, and especially if the tongue and lips were tremulous and the hands unsteady. Should sudden failure of strength occur, as may

happen at any period of the disease, brandy would be administered freely, and ether and strychnine, the latter especially would be injected subcutaneously. Exhaustion towards the end of a long and severe attack would be counteracted by similar expedients, together with the assiduous administration of concentrated liquid nourishment in small quantities. The patient may sometimes be carried through a period of extreme and almost hopeless danger by unremitting employment of one after another of these resources.

Symptoms of oppression of the nervous system not obviously due to exhaustion by protracted high temperature, particularly any tendency to heavy stupor, with small, weak pulse, would be an indication for the administration of two or three grains of calomel, unless the intestinal lesion appeared to be severe, in which case haemorrhage might result. On the sudden supervention of tympanites half a drachm or so of laudanum or liquor opii would be given. Complications would be dealt with as they arose—haemorrhage by means of some liquid preparation of opium or morphia to arrest peristalsis, by an ice-bag over the right iliac fossa, and by the subcutaneous injection of ergotin to tighten up the arteries and diminish the systolic output of the left ventricle. In case of perforation abdominal section has in a small proportion of cases saved the patient, if that were impracticable a large dose of opium together with morphia injected subcutaneously would offer the only chance.

ON A CASE ILLUSTRATING THE PROGNOSTIC SIGNIFICANCE OF THE BLOOD PRESSURE IN ACUTE RENAL DISEASE

Medical Society's Proceedings, 1888

It is well known that kidney disease is attended with increase of tension in the arterial system. This is most marked in the contracted granular affection of these organs, but it is present in almost every form of renal disease. I have, however, met with cases in which the arterial tension has been much below instead of above the normal average, and all such I have watched with unusual interest. I have twice seen low tension when the character of the urine, the age, antecedents, and condition of the patient have indicated cirrhosis of the kidney; in both the disease proved fatal with unusual rapidity. In acute renal dropsy I have found persistent pulse of low tension more frequently, and it has always been associated with an intractable character of the disease. A fatal case of this kind was the subject of a clinical lecture some years since.

In acute renal dropsy the artery is usually at first full between the beats, and rather large, but the beat is short and easily arrested. This corresponds to a period of temporary dilatation and weakness of the left ventricle. In the course of a week or ten days, however, the heart recovers itself, and the pulse acquires the character of moderate tension, which, in a case ending favourably, is sustained up to the time of complete recovery. I have learnt, therefore, to look for the supervention of a certain degree of tension in the pulse as an indication of favourable progress; but, as has been said already, in some cases it is missing. The defect of tension may be due to persistent weakness of the heart, the capillary or arterio-capillary resistance being present, but the driving power on which the degree of pressure in the intermediate arteries ultimately depends being deficient. That weakness of the central organ of circulation is of unfavourable prognostic import is easily seen, but the low tension is not always so caused; there is sometimes diminished resistance at the periphery; the capillaries and arterioles are relaxed, and allow the blood to slip through them as in pyrexia, and the pulse is not only weak but short. It

is not so easy to understand why this should be of bad augury, and it is only by observation that this conclusion has been reached.

The case to which these remarks lead up is summarised in the following account :—

J. L., aged 27, a carman, married and having three healthy children, of sober and steady habits, and previously in the enjoyment of good health, was admitted into St. Mary's Hospital on October 29, 1887, on account of acute renal dropsy. His mother, still alive, was much addicted to drink, which brought her to the workhouse, where she then was. There was no other unfavourable family history.

The patient was born and had lived all his life in London, and had had no illness since childhood, when he had smallpox. He underwent a certain amount of privation two years previously, when he was out of work for four months, but did not apparently suffer in health. For the last eighteen months he had been a carman. Without any special exposure to cold, he had four days previously noticed on rising that his face, arms, and legs were much swollen, and he had a bad cough. He remained at home for two days, and then returned to work, but on the day of his admission to the hospital the scrotum became swollen, and with this the face was pale and swollen, the eyelids puffy, and the extremities—the arms and hands more than the legs—affected by the characteristic firm, waxy oedema of early acute desquamative nephritis. The urine was found to have a specific gravity of 1030, and was almost entirely converted into a coagulum of albumen on boiling and adding nitric acid. It was straw-coloured, gave a very slight blood reaction with guiacum and ozonic ether ; no blood corpuscles were seen, but numerous epithelial casts. The temperature was slightly subnormal, the pulse 64.

I first saw the patient on November 1, and found the pulse 60, short and very compressible. The heart was normal in size, but the apex-beat could only just be felt, and could not be definitely localized, and there was no right ventricle impulse ; the sounds were weak and indistinct.

A week later the dropsy had everywhere increased, and there was a small amount of fluid in both pleural cavities. The pulse was still short, weak, and devoid of tension ; the heart somewhat enlarged downwards, the first sound short, the interval between this and the second rather longer than normal, the second sound weak. The amount of albumen in the urine was still very large, the coagulum occupying seven-eighths of the volume ; hyaline and granular casts were present.

The prognosis was at once formulated that the case would be

of long duration, the basis for this being the defective pulse-tension and weak blood-propulsion by the heart. It was at the same time considered probable that the large proportion of albumen was not due to any specially severe affection of the kidneys, but was a consequence of the languid circulation, which would almost permit of complete stasis in the capillaries ramifying between the convoluted tubes in the cortex of the kidneys, and, consequently, in the Malpighian tufts. It was anticipated, therefore, that if the circulation could be improved, the amount of albumen might diminish rapidly.

The patient was, of course, kept in bed. His diet was mainly milk. Tincture of iron with sulphate of magnesia was given at first, to which were added in a few days nux vomica and digitalis; dry cupping over the loins was practised.

At the end of a fortnight, on November 14, the dropsy had slightly diminished, but there was still much fluid in both pleural cavities. The pulse was short and compressible, the apex-beat of the heart just perceptible in the fifth space slightly outside the nipple line. The urine had a specific gravity of 1018, and the coagulated albumen occupied half its volume.

On November 21 a general improvement was noted in the pulse. The artery could easily be felt between the beats, and rolled under the finger, and was not so compressible. Simultaneously the amount of albumen had fallen, so that it was described as rather more than a trace. The dropsy and pleural effusion were much less.

On November 24 the apex-beat could be distinctly felt in the fifth space, three-quarters of an inch outside the nipple line. The first sound was here indistinct and rather prolonged instead of short, as before; the second sharp and louder than normal, the interval still much prolonged; the pulse was long, and not easily compressible; albumen one-eighth; dropsy nearly gone.

Two days later the patient appeared to be so nearly well that he was allowed by the resident surgeon to get up for a short time.

There was at once, December 5, some return of swelling in the legs. The apex-beat and heart sounds became weaker and the pulse more compressible, while the proportion of albumen rose to one-third.

That this was not an ordinary relapse due to chill was clear from the rapid return to the previous condition of the urine when he was sent back to bed. The explanation was that the heart was not equal to the maintenance of the circulation in the erect position, and with the languid movement of blood so caused came the increased amount of albumen.

He was now kept in bed. He took his food, slept well, remained free from dropsy, and had only a varying trace of albumen. Before it was considered safe again to allow him to rise he had an attack of tonsilitis, in which the temperature rose to 103·8° on January 23, and the urine was now of a dark purple colour, and contained four-fifths of its volume of albumen. Under the microscope however, there were scarcely any blood-corpuscles. The blood and albumen rapidly disappeared, and were succeeded three days later by uric acid crystals in large quantity. The attack was, in fact, one of haemoglobinuria. The patient is still in hospital, but his subsequent history possesses no points of interest.

The imperfect development of blood pressure to which I have called attention in this case is not to be looked upon as the cause of the slow recovery or of the complications observed in the course of the disease; it merely reveals the constitutional weakness to which they are to be attributed, and shows that the patient is made of poor stuff. In doing this, however, it fulfils a useful purpose, as it throws light on the prognosis. The occurrence of haematolysis and haemoglobinuria in the course of an attack of tonsilitis was an interesting confirmation of the opinion formed as to the feeble powers of resistance possessed by this young man's constitution.

The development or non-development of pulse-tension in the course of acute renal disease appears to me again to furnish some guidance in treatment. To raise the tone of the circulation is a help towards recovery when this is defective.

RENAL DISEASE AND THE CIRCULATION

Practitioner, 1901

THE Editor has imposed upon me the task of dealing with the relations between disease of the kidney and the cardio-vascular system, or, in other words, of discussing the effects of renal disease upon the circulation. This can be done only briefly and imperfectly in the space at my disposal.

The primary and dominant effect of disease of the kidneys on the motion of the blood is *obstruction in the capillaries and arterioles*. This appears to prevail in all the somatic structures, and it is no doubt provoked by the presence in the blood of nitrogenized waste which it is the office of the kidneys to eliminate. In what chemical form exactly this retained waste material exists we do not know fully and definitely, and the inquiry does not concern us here.

A question of great importance is whether the primary obstruction is in the capillaries, or by the stop-cock action of the arterioles. Notwithstanding the high authority of Dr. Oliver in all matters relating to the circulation, I am still of the opinion that the primary seat of obstruction is in the capillaries, and that the contraction of the arterioles is secondary to this. There is capillary repulsion as well as capillary attraction as a physical phenomenon. While water ascends in fine glass tubes, mercury almost refuses to enter them; the entire process of osmosis is governed by the affinities of the membrane and the fluid. Physiologically, according to my apprehension, the resistance to the onward movement of the blood in the capillary network determines the exudation of the liquor sanguinis from the capillaries into the interstices of the tissues, where it becomes available for their nutrition, which it is not while within the vessels. This resistance varies under normal conditions, and it is almost a corollary that it must be influenced in one or other direction by morbid states or constituents of the blood. The experiments of Sidney Ringer seem to demonstrate conclusively that resistance to the onward motion of the blood excited by substances introduced into it is entirely independent of the nervous system acting upon the arterioles.

In pathology, again, the retinal haemorrhages, which are so

common, appear to me to be incompatible with the idea that the resistance in the peripheral circulation which has determined the rupture of vessels so minute can have its seat elsewhere than in the capillaries. A similar conclusion is suggested by a study of cerebral haemorrhages. It is the minute arterioles which burst, and the obstruction must therefore have been beyond them.

Given the obstruction to the onward flow of blood through the arterio-capillary network, there is first a contraction of the small arteries which we assume to be protective—the stopcock action of George Johnson. The resistance being present, and all the channels through which the blood must pass being narrowed, if the blood is to circulate at anything like the normal rate there must be an increased driving power, which can only be supplied by the heart. The heart rises to the occasion, and contracts with greater vigour. We have thus, in the arterial side of the vascular system, resistance in front and increased pressure behind. There must consequently be high pressure in the intervening arteries from the aorta onwards, which is spoken of under various aliases—"high blood pressure," "high intravascular or intra-arterial pressure"; and since the internal fluid-pressure puts the arterial walls on the stretch, "high arterial tension," which, recognized in the radial at the wrist, becomes "high tension of the pulse," or "high pulse tension," and the like.

Applying now the above considerations to the simplest and most common illustration of the effects of renal disease on the cardio-vascular system, we may follow them as they are found to occur in the course of contracted granular disease of the kidney.

The Pulse.—Let us note first the characteristic pulse. What we feel in the pulse in our routine examination of it is not any expansion of the artery, any increase in its diameter, but, having more or less flattened the vessel by the pressure of the fingers during the interval, this change of shape is rectified by the increased internal pressure of the pulse wave, and this is what is felt. There being, then, the increased resistance in front in the arterio-capillary network, and the augmented pressure from behind, the artery does not allow itself to be flattened so easily, it remains full, and more or less tense between the beats. The special character of a renal, or of a high tension pulse is not the degree of pressure required to compress the artery, not the force or violence of the beat as it impresses the finger; these depend on the vigour of the heart's systole (and the impression may be singularly influenced by the size of the vessel); the point to observe is the fullness of the artery between the beats, the absence of the sudden subsidence of the vessel under the fingers as the beat passes.

The pulse, then, in the early stages of chronic Bright's disease, will usually be small, the artery being in a state of contraction. The beats will be inconspicuous from the small size of the vessel, and from the fact that it is not readily flattened on account of the internal pressure. Such a pulse is very often described as weak. It will be found, however, that the artery can be felt between the beats, and can be rolled under the finger, and, when the attempt is made to extinguish the pulsation and compress the artery, the pulse seems to become stronger as the increasing pressure is applied.

We may now trace the effects in the arterial walls of the increased internal pressure to which they are exposed, these effects constituting in a large proportion of cases the process by which renal disease proves fatal. The first is a physiological increase of the muscular fibres, so that the arteries may be able to control the circulation by dilating or contracting as may be required. The vessel can now be felt to be thickened when the blood is squeezed out of it as well as full between the beats of the pulse.

In course of time, notwithstanding the increased thickness of their walls, the arteries become more or less dilated by the distending force of the blood within them. There will be fibroid change also, as well as muscular hypertrophy in their walls, attributable probably to compression of the vasa vasorum, and consequent impaired nutrition. It is when changes of this kind have taken place that we have the characteristic "renal pulse" in its full development—large, the pulse wave long and dwelling on the finger, gradual in its rise and fall; or more sudden if the arterial system generally has undergone much degeneration; the artery, full between the beats, thick walled, rolled under the finger, like another tendon at the wrist, and followed with ease half-way up the forearm, usually tortuous. A pulse answering to this description is often sufficient for a diagnosis.

The Heart.—Pari or impari passu, with the changes in the vessels will have come changes in the heart. At first increased vigour of the systole with marked apex-thrust and accentuation of the aortic second sound. Gradually hypertrophy of the left ventricle, displacement of the apex downwards and slightly outwards, general systolic heave as well as distinct apex push. As in the case of the arteries so in the heart, fibroid change supervenes, and in the long run it depends on the relative progress of degenerative change in the vessels and heart—often, however, on quasi-accidental circumstances—whether cerebral arteries are ruptured by the pressure which the heart is able still to maintain, or the heart is beaten by the resistance in the circulation and undergoes dilatation.

One of the early indications of the latter occurrence is reduplica-

tion of the first sound, best heard to the inner side of the apex, denoting a failure of synchronism between the two ventricles in their systole.

As the changes in the heart and vessels advance the symptoms attending disease of the kidneys develop—headache, troubles of the digestion, loss of flesh, breathlessness, sleeplessness, irregular action of the heart, and in later stages, perhaps, attacks of nocturnal dyspnoea.

As is well known the form of renal disease under consideration is not as a rule attended with dropsy. Oedema, however, may come on at a late period, when it marks the supervention of heart-failure; or it may be determined at any time by an attack of bronchitis, or of influenza attended with cardiac asthenia, or by serious over-exertion or fatigue. There will always, when oedema sets in, be a decided increase in the amount of albumen in the urine, and the aggravation of symptoms is often set down to intercurrent tubular catarrh or to advance of the disease of the kidneys, whereas it is really due to failure of the heart.

The extraordinary and inexplicable cases in which attention is first called to the condition of the kidneys by sudden impairment of vision in a young and apparently healthy and vigorous adult, which is found to be due to retinal haemorrhages, have always in my experience exhibited marked high tension of the pulse, but advanced arterial change has not been traceable.

Acute Nephritis.—The relations between acute tubular nephritis and its effects on the circulation are not so easily followed. They are, indeed, extremely inconstant, and other factors besides the affection of the kidneys enter into the question. For example, when the nephritis is scarlatinal or consequent upon some other acute specific infection, the blood poison will have its own effect on the heart and vessels. There may also be pyrexia, which, even if slight, will relax the arterioles and capillaries, and exercise an influence on the action of the heart. The tendency to obstruction in the capillaries from the presence in the blood of nitrogenized impurities is, however, always present, though it may be more or less masked or neutralized by one or more of the influences enumerated.

Oedema is usually an early symptom in tubular nephritis, but there is no definite relation between the dropsy and the proportion of albumen present in the urine, nor is the dropsy always explained by the diminution in the quantity of urine passed.

The pulse is, in the early stages of this form of renal disease, rather small, the arteries being full between the beats. There is not, however, any marked tightening up of the artery, and on compression the wave is easily arrested. This last fact shows that

the heart is not acting with vigour, and on examination the impulse will be found to be diffuse and the first sound short. The heart has, in fact, failed to respond to the call made upon it by the increased resistance in the peripheral circulation, and instead of meeting it by hypertrophy, has become dilated. The obstruction has developed too rapidly for the heart to keep pace with it, and the heart has been at the same time subjected to debilitating influences.

The condition here described of the cardio-vascular system and pulse in the early stage of tubular nephritis has been dwelt upon because the prognosis of the cases depends greatly on the development of real arterial tension. So long as the pulse remains flaccid and the heart weak, there is no effort on the part of the constitution towards recovery. As the pulse becomes firm and long the amount of urine passed in the twenty-four hours increases, and the proportion of albumen diminishes, and the first step towards improvement is taken.

In chronic tubular nephritis, whether primary or consequent upon an acute attack, a moderate degree of arterial tension usually prevails, and it is not through cardio-vascular changes that a fatal termination is usually reached.

Amyloid disease of the kidneys gives rise to little increase of intra-vascular pressure. It is secondary to other diseases, which on the whole tend to relax the peripheral vessels.

The cardio-vascular changes resulting from renal disease not only give rise to consequences directly due to the lesions of the heart and vessels—the cardiac dilatation and asthenia with the attendant dyspnoea and dropsy, the cerebral and retinal and other haemorrhages from rupture of degenerate vessels—they play an important part in the production of phenomena which are considered to be direct effects of uraemic poisoning. Such, at any rate, is the case, in my judgment, with respect to uraemic convulsions.

Uraemic convulsions.—Convulsions are not produced by the experimental introduction into the blood of urea or any of its derivatives, or of any other of the constituents of urine. Convulsions do not necessarily occur in old standing disease of the kidneys, when the blood may fairly be supposed to be completely laden with renal impurities from imperfect elimination extending over years, when, indeed, the breath is charged with uraemic faetor. On the other hand, they may supervene at a quite early period of acute tubular nephritis when there has been no time for the accumulation of any large amount of uraemic matters. Some other cause of the convulsions must therefore be sought, and it seems to me that the explanation is to be found in stasis of the cortical cerebral circulation.

The intra-cranial pressure is governed by the arterial pressure, and must therefore be abnormally high in chronic renal disease. Retinal haemorrhages and optic ischaemia are not uncommon, and I have seen the sheath of the optic nerve distended into a bulb by fluid from the subarchnoid space just behind the globe of the eye, exactly as in cerebral tumour. The cerebral hemispheres must, when the intra-cranial pressure is excessive, be compressed against the vault of the cranium, and sometimes the convolutions are seen to be actually flattened. Whether from the degree of such pressure, as may be the case in contracted granular kidney, or from the heart being deficient in energy at the time when the resistance in the peripheral circulation sets in, as in tubular nephritis, it may happen that the onward movement of blood in the cortical arterioles and capillaries may be arrested. It is this arrest of the cortical circulation which, in my opinion, determines the attack of convulsions.

Convulsions of the most violent character may occur in association with extremely high arterial tension when there is no renal disease. The most remarkable instance, among several of the kind which have come under my observation, was the case of a medical man still living more than twenty years after the occurrence which I have related more than once. He presented himself one morning, saying laughingly that he had been sent by his wife, who said he had had some sort of fit in the night. His forehead was dotted with minute petechiae, and there could be no doubt that it was a convulsion that he had had. The tension of the pulse was extremely high. During the following night I was called to him, and found one convulsion following another, with intervals during which he was unconscious, and the respiration almost suspended. On the strength of the state of the pulse on the previous day—for it was now weak and intermitting—he was bled to thirty ounces. The last convulsion took place while the blood was flowing, and he made a rapid recovery.

It is well known that Cheyne-Stokes breathing is more commonly met with in renal disease than in any other association, and I have come to the conclusion that it is in virtue of the high arterial pressure. In my experience high pulse tension has been an invariable antecedent of the true rhythmical Cheyne-Stokes rise, fall, and suspension of the respiratory movement, whether or not there has been albuminuria. The question, however, cannot now be discussed.

Treatment.—There remain for consideration the therapeutical indications to be derived from the cardio-vascular conditions resulting from renal disease.

Of these the first is immediate and free venesection on the occurrence of uraemic convulsions. Sixteen or twenty ounces of blood should be taken at once, followed by a calomel purge. The advantage of free bleeding was forcibly insisted on many years ago by Sir Samuel Wilks. If a single withdrawal of blood do not stop the convulsions it may be repeated, and recurrent uraemic convulsions may be met by further venesection. In acute tubular nephritis bleeding on account of convulsions may be followed by immediate and remarkable improvement. Without going so far as to recommend venesection in this form of kidney disease, an expression of regret may be permitted that wet-cupping over the loins is now so little practised.

A further indication for treatment may be deduced from the fact that the damage to the vessels and heart, through which much of the suffering attending renal disease is brought about, and by which life is shortened, is due to high arterial tension. The reduction of the intra-vascular pressure ought to be an object continually in view. For this purpose the vascular relaxants have been tried—nitro-glycerine, the nitrites, the tetranitrate of erythrol. Unfortunately the effect of these substances is very fugitive, but the last named, which is slower and more persistent in its action, may sometimes be given with advantage.

The best means known to me of exercising a definite influence on unduly high intra-arterial pressure is through mercurial aperients. I am accustomed to attribute it to increased elimination, but, whatever the explanation, the clinical fact is undoubted. A dose of calomel, three to five grains, will often avert impending convulsions, or prevent their recurrence, will relieve the headache, stupor and twitchings, and may prevent uraemic paroxysmal dyspnoea in advanced kidney disease. The nightly attack of renal asthma I have seen prevented by 5 gr. of calomel, and recurrence obviated by small occasional doses. In the attack the arteries are tightened up to an extreme degree, and the asthma is essentially cardiac, the left ventricle being beaten by the resistance in the peripheral circulation. So also a single grain of pil. hydrarg. or hyd. c. cret., with rhubarb or colocynth and hyoscyamus, once, twice, or three times a week, according to the degree of tension in the pulse, exercises a favourable influence in the early stages of chronic Bright's disease, both on the symptoms and on the course of the disease.

In this brief and imperfect article I have been more concerned to bring out clearly the connexion between renal disease and the affections of the circulation resulting therefrom than to discuss the subject exhaustively.

A NOTE ON POSTURAL ALBUMINURIA

The British Medical Journal, 1904

FUNCTIONAL albuminuria, which was the subject of three important papers read before the Medical Society by the President, Dr. Rose Bradford, and Dr. West, and of an interesting debate, is a question which has occupied my attention for many years, and as time did not permit of my taking part in the discussion, I hope I may be allowed to make a few observations now.

The first point which comes out is the necessity for greater precision in the definition of what we mean by functional albuminuria. Albuminuria can never be physiological; the term "functional," though admissible in the sense given to it by Dr. Pavy as a contra-distinction to structural, is vague; the albuminuria of the apparently healthy, the designation accepted by Dr. Rose Bradford, is still more unsatisfactory. To begin with, many of the subjects of intermittent albuminuria are not in apparent health; they faint in church or at drill, as pointed out by Dr. Armstrong and Dr. Clement Dukes, and as a descriptive term it includes structural disease of various kinds, and continuous, as well as intermittent or occasional, presence of albumen in the urine. It can only serve as a starting-point for discrimination and classification.

Without entering upon this point, which occupied much of all three of the original papers, it may be said definitely that there is an affection which may be called postural albuminuria. We can eliminate early nephritis and nephritis in process of recovery, early stages of contracted granular kidney, unsuspected heart disease, albuminuria from fever, from overfeeding or indigestion, gout or oxaluria, or from adventitious discharges from different parts of the genito-urinary tract. There is left a group of cases in which the characteristic feature is albuminuria on rising from bed in the morning, usually passing off in the course of the day. These cases are exceedingly common, especially in boys and young men working up for examination, and, applied to them, the designation albuminuria of adolescence is perfectly appropriate, but the name postural albuminuria is no doubt better, as open to no uncer-

tainty. The albumen is not due to food; it does not appear if the patient remains in bed to breakfast, or after meals later in the day; it cannot, as was clearly shown by Dr. Rose Bradford, be toxaemic; it disappears quickly on lying down. It is obviously in relation with the erect posture after a night's rest in bed, and scarcely less obviously with imperfect cardio-vascular adjustment to the changed hydrostatic conditions. Albuminuria from excessive exertion is also circulatory, the cardio-vascular capacity for adjustment having been over-passed. It does not seem to me that any antecedent affection of the kidneys by febrile disease need be postulated.

The amount of albumen is usually small, but it may be very considerable. It is important that this form of albuminuria should be recognized, since the treatment required is the exact reverse of the milk diet and protection from exposure to cold and fatigue, which would be suitable in renal disease; and the debate will have been of signal service if it should lead to the adoption of the term postural.

The antecedent which in my experience has been most common is a neurotic family history, and the condition invariably existing in the patients themselves has been cardio-vascular instability. The pulse varies greatly in frequency and tension, often while under examination, certainly at different periods of the day, and is unduly influenced by changes of position, such as lying down or standing up. A distinctly high-tension pulse, not very uncommon in childhood and early youth, would take the patient out of the category of postural albuminuria, and would warrant suspicion of actual renal disease or of juvenile gout.

The action of the heart affords further evidence of the instability of the circulatory system. Both Dr. Pavy and Dr. Armstrong spoke of the violent cardiac impulse. According to my experience it is the impulse of the right ventricle which is forcible, the apex-beat being weak. Contrast between a weak apex-beat and left ventricle sounds and the powerful right ventricle impulse and loud sounds has seemed to me to be constant in these cases. Another observation of like significance is that the second sound is reduplicated when the patient lies down.

The prognosis is favourable. I have never known true postural albuminuria to resist treatment or to develop into actual renal disease. Usually all the treatment required is good simple food, fresh air, and vigorous exercise. There is no need to forbid school games, except perhaps house runs and long paper-chases. Tonics such as iron, arsenic, strychnine, and quinine will be useful in many of the cases. Constipation should be corrected by aloetic or other aperient of the tonic class. On the morning after a mild

pil. hydrarg. c. colocynth. et hyoscyamo the albumen may be absent even when habitually abundant. This should be borne in mind when a candidate for one of the public services has to undergo the medical examination.

If these cases are treated for renal disease, put on milk diet, protected from cold, and forbidden to take exercise, they will probaby go from bad to worse, and I have met with several instances of confirmed nervous valetudinarianism apparently attributable to this error in early life.

CASES OF KIDNEY DISEASE AND OF SUPPRESSED GOUT

FROM LECTURES ON "THE PULSE" IN THE *Lancet*, 1875, VOL. II

Kidney Disease

. . . I SHALL not occupy you long by illustrations of the association of high arterial tension with disease of the kidneys. The subject is one which I have considered here before, but it is strictly within the scope of this lecture to refer to cases in which the pulse has at once given the clue to the kidney disease. Most of you will remember the case I have already mentioned of a man, Henry C——, aged thirty-three, who applied for admission suffering from pain in the head, vomiting, and impairment of vision; he reeled and staggered also, exactly like a drunken man, and could not walk without support; his speech, again, was thick. He presented a complete picture of cerebellar disease. I sent him to the ward at once, with the remark that there was apparently disease of the cerebellum, probably, from his age and appearance, syphilitic, postponing examination till we could go into the case fully. When I came to his bedside, however, expecting the symptoms and history of an intracranial affection, I had scarcely placed my hand upon the pulse when my first-sight diagnosis fell to the ground. I saw I had to deal with renal disease; the urine was examined and found to be albuminous, the ophthalmoscope showed us albuminuric retinitis, instead of optic neuritis or ischaemia; the headache, the vomiting, the loss of vision were uraemic, and ultimately we had post-mortem demonstration of contracted granular kidneys.

In November, 1873, a former student of this hospital, one of our very best men, sent a clergyman to consult me on account of headache, attacks of giddiness, and impaired sight. Prepossessed with the idea of cerebral mischief, he had overlooked the contingency of renal disease, and had never examined the urine. Here, again, the pulse, long and hard, the artery standing out among the surrounding structures, at once suggested the diagnosis of kidney disease, which was confirmed by the presence of retinal haemorrhages, a clouded disc, and the familiar white spots round the disc and yellow spot. The diagnosis thus made was verified by the state of the urine, in which, however, at this time there was the merest trace

of albumen. Later the symptoms of Bright's disease became more marked, obstinate vomiting being prominent among them, and the patient died a short time ago.

I am reminded of another case in which I was called in consultation to a case of periodic vomiting in an old lady. Her pulse, small and weak, but with all the characters of tension, suggested an examination of the urine, which, as I had anticipated, was found to be albuminous. The vomiting was uraemic.

The examination of the urine is almost a matter of routine, but in both these cases it happened to be omitted. You are aware, however, that albumen is not always present in the urine in contracted granular disease of the kidneys; in the early stages it comes and goes, and later may occasionally disappear. You will see, therefore, the importance of having so clear an indication in the state of the pulse.

Gout and allied conditions of system as a cause of arterial tension.—The term "suppressed gout" is a convenient refuge for ignorance and uncertainty; it is accepted by the public as a sufficient explanation of any chronic or recurrent ailment, or, indeed, of anything and everything, and accordingly it is freely used by medical men. The expression, often spoken of contemptuously, has, however, considerable vitality, because it carries a certain amount of truth. Originally it embodied a conclusion formed from profound clinical observation, and, if it were not abused by indiscriminate application to all kinds of complaints, would be very useful and valuable.

In gout, as you are aware, the oxidation of nitrogenized waste, instead of going on to the formation of urea, has stopped short at uric acid, which accumulates in the blood and constitutes a poison to which the attacks of gouty inflammation are due. This, at any rate, is the theory which, if not demonstrably true in every point, is an excellent working hypothesis. But the destructive metamorphosis may fall short even of uric acid, and the blood may be contaminated by a variety of imperfectly oxidized matters. The presence of these impurities in the blood gives rise to the most varied consequences: frank acute gout, chronic gout with the formation of gouty concretions in the joints, on the ears, in the conjunctivae; the less honest forms of gout, pains in the toe, the heel, the loins, the head; attacks of vertigo, irritability of temper, loss of the faculty of attention, palpitation of the heart, cough and shortness of breath, dyspepsia, gravel or stone, inflammation or irritability of the bladder, eczema, and other skin affections. Besides these there are the so-called attacks of gout in the stomach or head, and sudden and extensive congestion of the lungs. After this list you will no longer wonder that everything is referred to sup-

pressed gout. There is one feature, however, common to the whole range of these affections—high arterial tension. Nitrogenized waste, at whatever stage of arrested oxidation, appears to provoke resistance to the passage of the blood through the capillaries and minute arteries, and this affords us the clue which guides us through the maze and gives precision to our ideas.

We are now dealing, as you will see, with the main cause of the two serious forms of disease we have already considered—degeneration of arteries and chronic granular contraction of the kidneys; and what makes it more important is, that at this stage we have the opportunity, not for palliative only, but for remedial treatment. The arterial degeneration is a direct result of the strain upon the walls of the vessels by excessively high tension. As to the renal disease, we cannot, as I have already said, speak so positively; we cannot say definitely whether it is produced by the state of the blood acting injuriously on the epithelium of the tubules, or whether it also is a consequence of high arterial tension.

The following are some of the cases I have seen which may serve to illustrate *different phases and stages of suppressed or spurious gout.*

I was called in March, 1873, to see a gentleman aged about 64, who was suffering from cough. He held a high position in the Colonial Service, and had spent a great part of his life in hot climates. He was supposed, on that account, to be susceptible to cold. He was stout, good-looking, and of very dignified appearance, but singularly stolid, dull, and uncommunicative. I permitted myself indeed, to make certain mental reflections on the kind of King Log sent out to administer our colonies. His symptoms, however, were not fully accounted for by the slight bronchial catarrh which was all I found on examination of the chest. More particularly my attention was arrested by a high degree of arterial tension, shown by the long, hard, labouring pulse of 84, and by the vessel remaining full between the beats. There were also the cardiac signs of high tension, a reduplicated first sound near the apex, and an accentuated aortic second sound.

By pushing my inquiries, I gradually learnt that my patient had for some little time been more easily fatigued than usual, had been often greatly flushed after dinner, unaccountably irascible, with lapses of memory at times, and, finally, it came out that in a letter written shortly before my visit there had been the most extraordinary anomalies of spelling, many words having been spelt backwards. Nothing could have been more significant than this train of symptoms associated with arterial tension; they were the premonitions of impending apoplexy; and apoplexy, as I learnt later, was the natural mode of death in his

family at about his age. The urine had a specific gravity of 1014, but had a good deep colour, and contained no albumen. The object to be attained, in order to escape the threatened danger, was to bring down the arterial tension. I gave calomel (three grains), with colocynth and colchicum, followed by a saline draught as a purgative, and iodide and citrate of potash with taraxacum; reduced the proportion of meat in the diet, substituting milk and fish, and for a time made dinner a midday meal; no restrictions were needed with regard to stimulants, the patient's habits being almost abstemious. This line of treatment was continued for some little time with the effect of removing all the unpleasant symptoms, and I found I had done my patient's mental power and social qualifications great injustice.

I saw little of him till February, 1874, when I was urgently called one evening, and found that, after similar warnings, he had partially lost power in his left limbs; the left side of the face was slightly paralysed, and the speech very thick and indistinct; there had been some mental confusion, but no loss of consciousness. A similar line of treatment was adopted, and now the dinner hour was permanently established at 2 p.m. Improvement again followed, but it was long before the face recovered itself, and, indeed, a slight inequality is still to be detected.

In February, 1875, my patient lost all appetite, and the tongue was much furred; there was a certain degree of tension in the pulse, but not so much in my judgment at the time as to warrant the free purgation I had previously employed. I gave aperients and bitter tonics without much effect. My patient fasted strictly through Lent, and lost considerably in weight. After Easter I was asked to see him on account of slight bronchial catarrh. While recovering from this he was seized with severe influenza; and just as he seemed to have shaken off this, and before he left his bedroom, a sudden and violent attack of congestion of the lungs supervened, which nearly proved fatal within twenty-four hours. My treatment of the last complication was dictated by my previous knowledge of the case, and was of a kind I should not have ventured to adopt without it. The congestion of the lungs was simply another consequence of the blood-contamination, of which the arterial tension had formerly afforded evidence; but the tension had lately been less manifest in consequence of weakness—had, in fact, been disguised. Recognizing this, I resorted as before to very free purgation, and relief came when this was obtained.

Here we reach one of the most interesting points in the case. The convalescence was not satisfactory; the temperature sub-

sided, there was free and loose expectoration of rusty muco-purulent matter, but the appetite did not return, the tongue did not clean, the pulse was weak but long, and there was no recovery of strength till one day there was pain in the ball of the great toe. A mustard poultice was applied, and the patient had his first attack of gout, which was perfectly characteristic, and, considering the circumstances, remarkably severe. After this he was well almost at once, and remains well up to the present time.

Here residence in a hot climate, a vegetable diet, and abstinence from the heavier wines, had prevented the full development of gout in a constitution strongly predisposed by hereditary tendency; but a return to this country and to English habits of life, the consumption of more animal food, and the diminished cutaneous excretion, had speedily led to the accumulation in the blood of imperfectly oxidized nitrogenized waste and its consequences. The truth of the inferences I had drawn as to the danger arising from the arterial tension, and as to the cause of this tension, was fully established by the course of events. The occurrence of a first attack of gout after the treatment pursued was remarkable, and indicated, in my opinion, that the metamorphosis of nitrogenized matter had stopped short of the formation of uric acid.

Cerebral symptoms.—I have had another interesting case under observation recently, which I may briefly relate. A medical man in the country returned home one day last April, about 2 p.m., from an unusually long and cold ride. He took some soup and a glass of sherry, and found, as he thought, that the wine got into his head, for he felt giddy and had to lie down; his speech was thick; he was violently sick; and it was some little time before he could proceed to the remainder of his day's work; and when he tried to walk it was noticed that his left limbs had lost power in some degree; they felt numbed, also, at first, and later were in a state of hyperaesthesia. One medical friend said it was "biliousness"; another thought more seriously of his condition, and he came to town to consult me a week after the attack.

He was the youngest of a healthy family of sixteen—healthy, that is, except as to a strong gouty tendency. He had a large country practice, was of active habits, worked very hard, was abstemious both as to food and alcoholic drinks; but notwithstanding this he had become decidedly stout, and had from time to time had gouty symptoms. The vertigo and sickness might, of course, have been due to gastric or hepatic derangement.

had they been the only symptoms; but the loss of power and sensibility and subsequent hyperaesthesia, and the thickness of speech, remains of which existed when I saw him, were significant of some cerebral mischief; and the pulse gave evidence of very high arterial tension, being hard, long, and rather small. Here, rest from worry, mercurial purgatives, potash salts, a farinaceous and fish diet, with weak spirit-and-water, or water only, as drink, have resulted in an effectual lowering of the vascular tension, loss of redundant fat, and removal of the symptoms. A too rigorous application of the principles laid down, indeed, led to attacks of giddiness from imperfect supply of blood to the brain; and I found it necessary to recommend citrate of quinine and iron for a time, with iodide of potassium.

Sleeplessness.—In the following case, I have no special reason for saying that there was gout, but the patient was, I believe, on the verge of apoplexy. He was 77 years of age, but remarkably hale and vigorous, and would have passed well for 60. He had been suffering for six weeks from sleeplessness and a feeling of extreme restlessness; naturally, also, he felt weak and worn in consequence. Tonics had been prescribed, but had not suited him; and he had taken chloral and bromide of potassium, obtaining by their means an occasional night's rest but no efficient relief. His appetite was good and the bowels regular—indeed he rather prided himself on his management of the bowels; he complained a little, however, of indigestion. I found in the pulse evidences of an extraordinary degree of tension, together with senile change in the vessels, though less than might have been expected; the artery large, cord-like, slightly uneven, firm, capable of being rolled under the fingers, and it could be followed half-way up the forearm, the pulsation being long and strong. Nothing could be more full of danger than this condition at the time of life. I gave, therefore, purgatives more freely than usual. After three or four doses the evacuations became so frequent and free that I feared I had carried the treatment too far. I found, however, that they were not liquid, but simply extremely copious, soft, dark stools; there had evidently been great faecal accumulation, notwithstanding my patient's assiduity in his attention to his bowels. It was some little time before his system regained its equilibrium; but the tension was quickly reduced, and the restlessness passed off. I attributed the sleeplessness to the high blood-pressure, which overcame the tonic contraction of the cerebral arteries, by which the anaemic condition of the brain essential to sleep is secured. Persistance of this degree of pressure must, I think, have resulted in rupture.

Nocturnal Dyspnoea.—But apoplexy is not the only danger to which the arterial tension of retained nitrogenized waste gives rise. The heart may cease to be equal to the struggle against the increased resistance and become dilated, especially if there be degeneration of its structure. You know the usual consequences of this condition: breathlessness on exertion, then cough, with paroxysmal and habitual dyspnoea, oedema, general dropsy and all its miseries. In an early stage, with shortness of breath and cough, it is not uncommon for dyspnoea to come on at 3 or 4 a.m., compelling the patient to sit up, and perhaps pass the remainder of the night in this position. This speedily leads up to further symptoms; but when it depends on arterial tension, as is often the case, it may be relieved by treatment which removes this condition.

I have now under observation a gentleman, aged 78, who, notwithstanding his years, is gradually recovering (has now, September, quite recovered) from severe nocturnal dyspnoea. He is crippled by chronic gout, has a well-characterized senile pulse, and a weak heart. The treatment has been a free resort to aperients and a cautious administration of digitalis and iron. In the case of a lady aged 64, first seen in January of this year, who suffered greatly from this form of dyspnoea, together with harassing cough, complete relief was afforded by the removal of arterial tension by similar means. These cases are worthy of relation in detail had I the time, and I have notes of many others equally worthy of your attention: one, for example, in which I have in the course of 6 or 8 years watched the gradual development of disease of the aortic valves under the influence of strain from arterial tension in a gentleman, the subject of gout. In another case, sent to me from the country in May, the patient had suddenly become liable to dyspnoea, attended with lividity of face on the slightest exertion; the mode of life and clinical history having been exactly that of imperfect nitrogenized elimination and arterial tension. This gentleman had been seriously injured by Bantingism.

I can only mention these few illustrations; but I may put in a few words the lesson to be learnt from them. It is, that when the heart shows signs of weakness, and especially when there is reason to believe that there is dilatation of its cavities and degeneration of its muscular walls, if there is any degree of arterial tension, the most effectual relief you can afford is to diminish the peripheral resistance. This can best be done by aperients, and by none so well as by pills containing one or other of the mercurial preparations; afterwards iodide

and citrate of potash, or iron and digitalis, or tonics may be given. There is often a degree of debility which seems to contra-indicate purgatives altogether; but it will be found that the powers are oppressed rather than depressed, and that the weakness is apparent rather than real. Tonics in these cases only do harm until after the action of purgatives and other eliminants, when also digitalis may find its opportunity; but frequently, when you cannot strengthen the weak heart, you can give it less to do, and so make it equal to its work.

I proceed to illustrate *other effects of retained nitrogenized effete matters* with peripheral resistance to the circulation and high arterial tension.

Palpitation.—A barrister, 35 years of age, consulted me in November, 1874. He was beginning to succeed in his profession, but his prospects were threatened by his state of health. After any heavy work, or an appearance in Court, he had palpitation of the heart and throbbing in the neck and head, with a feeling as if he were about to faint, rendering it necessary to take some stimulant; he would also be entirely sleepless at night. This had been attributed to weakness, and he had been taking meat three times a day rather largely at each meal, with a liberal allowance of sherry. Notwithstanding this his symptoms increased upon him. His appetite was good, his bowels regular, but the tongue was white and indented. I found the pulse full, long, and hard, and the aortic second sound unduly loud, and took these as my indications for treatment. I gave him a gentle aperient pill for four nights in succession, and afterwards every second night. In addition, I ordered phosphate and carbonate of soda, with tincture of ginger in infusion of calumba, and revolutionized his diet, allowing meat and wine (claret) only once a day; breakfast and lunch to consist of milk and farinaceous food. He got well at once.

In the last case there were no specially gouty family antecedents, and the choking of the blood with nitrogenized waste was a direct result of the mode of life. When there is hereditary tendency to gout, imperfect metamorphosis and elimination of nitrogenized matters occur much more readily, and sedentary habits, without any mistaken system of diet, or very moderate indulgence in beer or the stronger wines, with a liberal, but not excessive, amount of flesh meat, will, in spite of an active out-door life, result in the development of a gouty or pseudo-gouty state of system. I have been astonished to see how early in life arterial tension is met with in the children of a gouty parent. I have found it well marked at twenty-one, and seen prominent and tortuous temporal arteries at twenty-four, associated with unaccountable feelings of weakness,

weariness, and depression, headaches, loss of application and of interest in the ordinary pursuits and enjoyments of life, and other like symptoms. I will not give these cases in detail, but, instead of them, the following, which illustrates the same point.

Lassitude.—A young surgical friend, about 32 years of age, consulted me on account of lassitude, inaptitude for work, loss of energy, headache, and other vague symptoms. I gave him the advice we always give each other—that is, to take a holiday. He took one and was better. By and by he came back again with the same story, and he was now uneasy about his heart and lungs, had morning cough, and was sooner out of breath than usual. He could not always be going away on holiday, and I went more carefully into his case. He was remarkably strong physically, but had not quite the colour he ought to have had, took less exercise perhaps than formerly, but still played cricket. His appetite was good and bowels regular. There was nothing particularly wrong that I could see till I placed my fingers on the pulse, when I found a largish cord-like artery standing out among the other structures, and traceable far up the forearm, the pulsation, of course, long. It instantly flashed upon me that his father was the subject of old-standing gout; the cause of his ailments was clear, fortunately the cure also. I recommended the treatment with which you must now be sufficiently familiar, and forbade my young friend his beer. In a week his arterial tension was reduced, and the symptoms were gone.

Treatment.—It is time now that I should say something more definite of the treatment you have seen me so often recommend, and give my reasons for adopting it. The aperient pills are sometimes the ordinary calomel and colocynth, or blue-pill and colocynth or rhubarb pills, with perhaps hyoscyamus; sometimes colchicum or ipecacuanha is added. One or two pills will be taken nightly for two, three, or four consecutive nights, or on alternate nights, or twice a week, followed in the morning by a saline draught—sulphate or phosphate of soda, a Seidlitz powder, a dose of some aperient mineral water or of white mixture. The strength of the purgative and the frequency of its repetition will depend on the urgency of the symptoms, the degree of constipation, and many other circumstances. At the same time I have generally given a mixture containing potash salts; the iodide, two to five grains; and citrate, fifteen to twenty-five grains, with spirit of ammonia and some vegetable bitter, as taraxacum, calumba, hop, gentian, or the like; or, instead of potash, soda salts, the carbonate or phosphate in a similar vehicle. The object in view is the reduction of the arterial tension, and this is effected to some extent by purgation as

such, which withdraws a certain amount of fluid from the blood, and relaxes the vascular system of the gastro-intestinal mucous membrane. But while any purgative will do so much, the effect will be imperfect and temporary unless the nitrogenized waste, which is the cause of the obstruction in the capillaries and arterioles is eliminated.

Now the liver is the great organ and instrument of metamorphosis; urea and uric acid are believed to be formed here, and on the efficiency of its functional action certainly depends the due elimination in the urine of the products of the disintegration of nitrogenized compounds. Mercury has long been supposed to act specially on the liver, and though experiments on animals have appeared to negative this idea, it is matter of observation that mercurial purgatives bring away darker and more bilious stools; it is also matter of observation that they produce a more marked and lasting effect on undue tension in the arterial circulation. I was taught as a student that other purgatives would do all the good which could be obtained from blue-pill and calomel without the risks said to attend the administration of mercury, and for a long time I was altogether sceptical when people who called themselves bilious insisted on the superiority of their favourite blue-pill. After a time, however, I was compelled to admit that the mercurial purgatives afforded a relief which no others did, but I only understood it when I began to observe that they had a greater effect on arterial tension. At present we must take our stand simply on the clinical fact that purgatives containing some preparation of mercury have this effect of lowering arterial tension. The explanation—namely, that this is produced through the influence of the metal on the liver conducing to a more perfect metamorphosis of nitrogenized matters—we hold less firmly. We may, if we prefer it, trace the good results to removal of bile from the upper part of the small intestine, which would otherwise have been reabsorbed and again separated from the portal blood by the liver.

The potash salts are given as eliminants, their effect in this respect having been abundantly demonstrated. Diet is of the greatest importance. Meat should be consumed sparingly, its place in supplying nitrogenized food being to a great extent taken by milk and fish. Soups are forbidden. Little alcohol should be taken, and only in the form of the light wines or freely diluted spirit. Water, free from lime salts, should be drunk in large amount, better apart from meals.

We have by no means exhausted the subject of gout, pseudo-gout, suppressed gout, and allied conditions. I have mentioned *lead poisoning* as a cause of arterial tension—it is in effect a cause

of real subacute or chronic gout, with all its attendant evils: chalk-stones, deformity of the hands and feet, contracted granular kidney, heart disease, arterial tension and degeneration of the large and small blood-vessels, apoplexy, etc. It is, of course, among hospital patients, painters, and other workers in lead, that gout from lead-poisoning is met with, and they have furnished some of the most terrible examples of gouty disease I have met with. There is a "gout pill" which these artisan classes obtain from chemists, which appears to be most effectual in suppressing gouty paroxysms and pain; there is not, therefore, the check on habits tending to produce gout which the paroxysms would impose, while the poison, never eliminated or destroyed by attacks of acute gout, accumulates in the system in an extraordinary degree.

I have seen several cases of *gangrene of the lungs* in individuals saturated in this way with gout, and believe the lung affection to have been due to this condition of system, in which there is apparently a tendency to thrombosis in the pulmonary vessels. It is interesting and suggestive to note that in the case of gangrene of the lungs recently under my care in the hospital we found high arterial tension. The patient, an ostler, was only thirty-eight years of age, had enjoyed good health, said he had been temperate, had not had gout or rheumatism, and was not the subject of lead-poisoning. He died on June 5, the day after his admission, and the gangrene was found to be due to thrombosis in the pulmonary artery. It would thus appear that the state of blood which leads to thrombosis gives rise to high tension.

CASES OF HYDROPHOBIA

Clinical Society's Transactions, 1883

I DESIRE to submit the case to be described to the criticism of the Society before it passes into circulation as one of recovery from hydrophobia.

The patient, a boy, named John Harris, aged 12, was admitted into St. Mary's Hospital about 5 p.m. on February 25, 1876, suffering from violent convulsive attacks, which had been going on for two days. I happened to arrive in the ward at the same time, and my attention was immediately called to the case. At first glance I took the condition to be that known as the status epilepticus, but when the convulsion passed off the boy was found to be perfectly conscious and in full possession of his faculties. During the intermissions, which were very short, violent simultaneous jerkings of all the limbs and of the body were observed, most marked in the legs. In the extremities the jerks were sudden movements of extension, in the body they were opisthotonic. Frequent heavy sighing also was noticed. The paroxysms, when closely watched, were ushered in by a loud and deep breath, the head and shoulders were thrown back, and the body was extended as in commencing opisthotonos; then most violent and rapid rotatory movements of the head and neck set in, which were accompanied by equally rapid inspiration and expiration, with laryngeal sounds not certainly like the bark of a dog, but requiring only a little imagination to suggest the resemblance. After the paroxysm, which lasted one or two minutes, the patient moaned and complained of pain in the head.

His parents, both of whom came with him to the hospital, stated that he had been perfectly well till the morning of the 23rd, when he did not eat his breakfast and seemed to be ailing. At about 12 o'clock on that day he was seized with the first "fit," as the paroxysms were called, and in it fell down some steps; the fits had continued up to the time of his admission, gradually becoming more severe. There was no horror of water, but the patient was unable to swallow. Fluid was offered him first in a feeder, while he was lying down, afterwards from a mug when in the sitting

posture; he accepted it willingly, but the attempt to swallow, or rather the arrival of the fluid in the mouth, immediately brought on a spasm, which was more violent in the experiment of drinking in the ordinary way from an open vessel. Later it was found that contact of a cold object with the skin, placing the hand gently over the heart, the sound of water falling into a basin, or the approach of water, also induced the attacks. An attempt to examine the fundus of the eye by the ophthalmoscope also provoked spasm. The countenance was anxious, the face pale, skin cold and clammy. Temperature normal. Pulse 108, small, weak and hesitating. Respiration of deep sighing character. Slight increase of saliva noted.

The symptoms were such that the idea of hydrophobia at once suggested itself. On inquiry from the boy's parents they knew of no dog he could have been in contact with except one which was still alive and well. The boy himself, when questioned, mentioned only the same animal. Asked if he had played with any dog now dead, he gave the following history:—He and two other boys had found a stray dog which they detained; it snapped at several children and bit him and another boy, whose name or whereabouts he did not know, upon which, having been told it was mad, they drowned it in a pail of water. He showed first his left, then recollecting himself, the right hand as the part bitten, and at the point indicated in the web of skin between the finger and thumb there was found a small mark as of a puncture, with an induration in the skin resembling that of a small hard chancre. It was slightly tender. The parents then recollected the circumstance, but neither they nor the boy had attached any importance to the bite, or had connected it with the existing symptoms. At this time no date could be obtained, but the occurrence had taken place since Christmas.

Nitrite of amyl was first administered by inhalation. Two doses of five drops had no effect, and at 7 p.m. chloral 20 gr., brandy 1 oz., and beef jelly 2 oz. were given as enema.

The convulsions ceased, and the boy slept, perspiring freely. At 10 p.m. he felt better, and was when awake quite sensible. Pulse 112, temp. 98·6°. The enema was repeated at this time, and again at 1 a.m. on the 26th. At 3.20 there were two or three strong spasms, whereupon the enema was again given. At 4.30 he had slept soundly, and had no more convulsions. He drank some milk, the first fluid he had swallowed for thirty hours. Brandy and beef tea given by the rectum. Asleep till 7.45, complained of pain in head and abdomen. Water applied to the face brought on a violent spasm. At 1 p.m. the pain in the head was so severe that a cold lotion was applied; this caused an immediate return

of the spasms; hot water did not have the same effect and gave great relief, so that the boy begged to have it continued. The hand placed over the heart induced spasms. Pulse 104, temp. 98·8°. No ulcers or vesicles found upon or under the tongue. 10 p.m.—Occasional slight spasms. The boy has eaten bread and butter, and drunk a little milk. At times the breathing oppressed. Pupils large. Pulse 100, temp. 98·8°. Beef tea and brandy enemata had been given every three hours during the day. The chloral now again added. February 27. A good night. Occasional slight spasms, but had been able to drink milk freely without distress and ate an egg and bread and butter for breakfast. No spasms, even when washed, but a violent convulsion followed slight pressure with the hand over the heart.

He was apparently well on February 28, but at 11.30 a.m. on February 29 there was severe headache and slight return of convulsions. Chloral 10 gr. was given on which the spasms ceased, though the headache continued. He had rabbit for dinner and enjoyed it, and was pretty well up to 9.30 p.m., when the spasms returned in very violent form and were almost continuous, they seemed to cause intense pain in the head. Chloral 10 gr. was given by the mouth and half an ounce of brandy in milk. At 10 p.m., the spasms continuing, chloral 20 gr. was given by the rectum with beef tea and brandy, and again at 10.30. At 11 p.m., the boy was dosing, but there were still spasms. He had a good night, but in the morning was very pale and had headache. His tongue was dry, more convulsions at 10 a.m., so chloral 20 gr. was given by the mouth. At 2.30 p.m. the same dose was given by the bowel with brandy and beef tea, and at 3.30 and 9 p.m. 10 gr. were given. Minute vesicles were seen on the under surface of the tongue near the tip.

After this formidable relapse it was considered more prudent to give chloral 10 gr. and brandy regularly three times a day, and they were continued till the 11th of March, the boy being up and going about the ward. The boy remained in hospital till April 2. The first time he went to the hospital chapel he was thrown into a state of uncontrollable excitement by the notes of the organ and had to be carried out, and he could not bear the sound of the street organs. He was kept under observation for some time after his discharge, and had no return of convulsions, but he was much more excitable than before the attack, so that he could not return to school.

The notes of the attack are placed before the Society, just as they were written six years ago. It was my opinion at the time that the attack was one of hydrophobia, and the only reason for

modifying this opinion would be the recovery of the patient. Had the boy died the diagnosis would never have been questioned. The hypothesis of simulation, whether wilful or as the result of nervous excitement set up by the bite of a dog, may be entirely set aside. The idea of hydrophobia had suggested itself to the parents, but even with this in their minds they had not been able to make out any history of a bite. From the boy himself we could learn nothing until, after plying him with every other kind of question, he was asked if he had ever played with a strange dog; and when his recollection was thus prompted he was at first mistaken as to the hand which had been bitten. Imagination, therefore, had played no part in the production of the symptoms. Tetanus, again, may be excluded, as although some of the spasms produced momentary opisthotonos, the essential character of the paroxysms was clonic and not tonic. The only remaining hypothesis which need be considered is that the attacks were epileptic or allied to hysteroepilepsy in character. Against this must be set the fact that there was no loss of consciousness during the paroxysms or stupor after them, that his mental condition was free from any hysterical excitement, and his conduct and talk quite natural. Very strong evidence in favour of the attack being true hydrophobia was furnished by the state of the cicatrix of the bite, which, as has been stated, was indurated and tender.

The following cases, which have been under observation since, are related by way of appendix.

Case 2.—Alice R., aged 13, a bright, healthy little girl, was bitten by a strange cat on the forefinger, June 28, 1881. The wound was dressed at the hospital, but inflamed, and did not heal for some days. She was afterwards quite well till July 30, when she complained of nausea, sickness and loss of power in the arms; she had a restless night, and next day had no appetite, but was thirsty, and gradually became excited. She was admitted to hospital about 10 p.m. on the 31st, when she was restless, excited, delirious, but could answer questions. She asked for water, but when it was brought said she could only take it from a spoon. Even then she hesitated, and at length opened the mouth wide and tossed the water in. She swallowed some of it, but with much difficulty, and the attempt excited spasm of the pharynx and neck. The mouth was dry and clammy, and she frequently hawked up viscid mucus, but did not spit it out. During the night she became rapidly worse, restless, delirious, talking incoherently, in terror when any one approached her, and she was soon quite unable to swallow. She died exhausted at 9.30 a.m., about 12 hours after her admission.

On examination five hours after death by Dr. Henderson no marked naked eye changes were present in the meninges, cortex, and substance of the brain, and cerebellum. In the floor of the fourth ventricle were a few spots of bright red congestion. The veins of the spinal cord were everywhere full, and much clear serum escaped when the spinal dura mater was opened. In the cervical region there was hyperaemia of the cord, not elsewhere.

Sections from the convolutions, pons varolii, and cord showed congestion of vessels and a few punctate extravasations into the perivascular sheaths, but no cellular infiltration.

CASE 3.—Pseudo-hydrophobia; death.—H. H., aged 26, was admitted October 25, 1876. He had been unfortunate in business in the North, had had much anxiety, and had taken rather to drink. Coming to London he had been engaged as a private policeman, and had had much excitement. Five years before he had been bitten by a dog on the ball of the thumb, and inflammation extending up the arm had resulted. He was said to have thought nothing of it till three days before admission, when after giving evidence in Court he went in a state of great excitement to take a glass of spirits. As far as could be ascertained some of it got into the larynx and provoked violent spasm, upon which he began to tear at his throat and say he was mad.

He was sleepless and excited from this time till admitted, and had then an anxious expression of countenance and most excited manner. He dreaded the approach of liquids, and when he tried to swallow there were spasms of the muscles of the pharynx and neck, gasping for breath, much exclamation, and a look of dread and horror. He could swallow solids. The mouth, fauces and pharynx presented nothing abnormal. The pulse was irregular, the temperature 100·6°. When I spoke to him firmly the patient was able to control himself and to answer questions clearly and calmly. Beef tea, brandy, and chloral 20 gr. were ordered to be administered every three hours by the rectum. During the afternoon the excitement persisted; the patient would jump out of bed saying he was in a yellow fog and could not breathe, waving his arms to keep nurses and others away, trying to get the fog out of his throat with his fingers. He said he was going to be murdered. He was seen by me several times in the course of the day and could always be quieted, but in the evening it was found necessary to remove him to an isolation ward, after which he was quieter, and was able to swallow milk.

During the night he had some sleep, but was noisy at times, tried once to strangle himself, and also to get out of the window, but in the morning was better and less agitated. Pulse

80, intermittent, temp. 100°, mouth dry. He complained of oppression in breathing. He took bread and milk from time to time during the day. Varied much but was on the whole more quiet. At 5 p.m. I was called to see him after lecture, and found that his wife and child had been allowed to see him, upon which he became wildly agitated, bidding them goodbye, reproaching himself for bringing trouble upon them, saying he must not kiss the child, etc. He was now utterly uncontrollable, and had to be secured in bed; he hawked up and spat out viscid mucus, clutched at bystanders, shouted and exhibited every form of violence. It was impossible to administer the enema, and chloroform was given, and while under its partial influence he swallowed and breathed without difficulty. He again became violent and died exhausted about 11 p.m.

At the post-mortem examination, 15 hours after death, the rigor mortis was very marked. The blood was dark and liquid, the lungs extremely congested with frothy mucus in the tubes, the kidneys also intensely congested. Brain—meninges injected but transparent, the veins turgid, cortical grey matter dark, white substance greatly congested, no embolism. Spinal cord, vessels engorged and substance congested.

This case shows that the reflex excitability of the pons and medulla may be exalted to the point of simulating hydrophobic spasm by emotional excitement, which presumably has its seat in the higher centres, the cerebral hemispheres, and other instances are not wanting which confirm this as a possibility. With regard to the lesions observed in hydrophobia, I look upon them not as the cause of the symptoms, but as the structural damage done by functional disturbance of extreme violence.

ON THE PHYSIOLOGY OF THE ACT OF VOMITING

Practitioner, 1875

In the interesting article on vomiting in the *Practitioner* for January, by the Editor, an active part in the expulsion of the contents of the stomach is attributed to the diaphragm ; that is, the stomach is said to be compressed by a simultaneous contraction of the diaphragm and abdominal muscles. This, moreover, is what is taught in most of the standard works on physiology, and it is considered to be established by an experiment in which the contents of the stomach were expelled by the contraction of the diaphragm after the abdominal parietes had been cut away to near the linea alba.

We cannot attach demonstrative value to an experiment in which such extensive mutilation is practised, and I have been led by observation of the phenomena of vomiting to question the accuracy of the generally received account of its production, and to come to the conclusion that the contraction of the diaphragm does not coincide in time with that of the abdominal muscles, but that the diaphragm first descends and is fixed by closure of the glottis, forming thus a firm but passive and merely resisting plane, against which the stomach is pressed by the actively contracting muscles of the abdominal wall.

I might cite in favour of this view the anatomical consideration that the oesophageal opening in the diaphragm lies between the decussating fibres of the pillars, and that when these fibres are contracting powerfully they will compress the oesophagus. This point, however, has been frequently set forth, and all *à priori* arguments, even from anatomical facts, must yield to actual observation.

I have attempted direct experimental observations, but under circumstances which did not permit of definite conclusions, since they were made on animals in which the anterior wall of the thorax was removed for the purposes of another investigation. Such as they were, however, they were confirmatory. But I must rely on what may be noted in the human subject in the act of vomiting. During the antecedent nausea and in the intervals of the paroxysms there is shallow respiration with a wide-open glottis, together with

frequent sighing. A paroxysm is ushered in by a sudden respiratory gasp—that is, by a descent or contraction of the diaphragm—and the success of the effort of expulsion appears to depend greatly upon the extent of this descent, and upon the point at which the diaphragm is arrested and fixed by the closure of the glottis. If the diaphragm were in active contraction at the moment of actual vomiting, we might expect that at any rate some of the sounds then produced would be inspiratory, whereas they are entirely expiratory, and much air is often forced out of the chest by the same act which expels the contents of the stomach. Any contraction of the diaphragm must thus be overcome as well as the resistance of the glottis.

Another consequence of contraction of the diaphragm as a part of the expulsive effort in vomiting would be a tendency to inspiration of the vomited matters while passing over the orifice of the larynx, which we might expect to see abundantly realized from time to time; whereas any slight entry of these matters which takes place, occurs in the inspiration which follows the actual vomiting. This same deep inspiration, again, after the convulsive retch, would appear to be conclusive evidence that the diaphragm had no share in it. It may be added that we should scarcely have the powerful compression of the lungs and heart in the act of vomiting, of which we sometimes seek to avail ourselves therapeutically, were the diaphragm contracting entirely against the expiratory muscles. Finally, the expulsive effort may be defeated by maintaining the glottis open. Such, at least, is my conclusion from a painful personal experience or experiment carried to the extent of my powers of endurance.

This note may be fitly ended by an observation in which the theory of the act of vomiting here maintained was turned to practical account. While resident medical officer to St. Mary's Hospital I performed the operation of tracheotomy on a young child. Two or three days afterwards I was called to the child, and found it in convulsions. Watching it carefully, I perceived that the general convulsive movements were preceded by violent contraction of the abdominal muscles, with facial movements suggestive of vomiting. The attempt to expel the contents of the stomach was, however, defeated by the absence of *appui* for the diaphragm by closure of the glottis, due to the opening in the trachea. Recognizing this, I closed the tube at the moment of each expulsive effort; free vomiting occurred, and the convulsions at once ceased.

(Intubation of the larynx will prevent vomiting, such as the obstinate vomiting of pregnancy.—Ed.)

ON DISPLACEMENT OF THE BLADDER AS A CAUSE OF TEDIOUS LABOUR

Transactions of the Obstetrical Society of London, 1864

I VENTURE to bring before the Society one or two points in obstetric practice, relating chiefly to some causes of protracted first stage of labour, the results of observations made while I held the office of Resident Obstetric Officer at St. Mary's Hospital in 1858–9. They were intended for publication at that time, in two papers, one "On Displacement and other Abnormal Conditions of the Bladder as a Cause of Delay in the first Stage of Labour," the other "On the Influence of Position on the Progress of Parturition," but the latter subject has since been under discussion before this Society and elsewhere, and I shall consequently treat of it very briefly, the former, however, I shall go into more fully, as I do not find in the systematic works on midwifery, which I have had the opportunity of consulting, or in the "Obstetrical Transactions," any complete account of the disturbing influence which derangement of the bladder seems to exert on the first stage of labour.

Undue distension of the bladder, prolapsus with accumulation of urine in it during labour, have long been known as sources of danger, and causes of pain and delay; and especially prolapsus with distension, as a mechanical impediment to the passage of the child's head, and as endangering the integrity of the organ, has received the consideration it demands, but I have been led to conclude that displacement and other conditions of the bladder, without involving the same serious consequences, frequently cause labour to be tedious and painful in the first stage, interfering with the dilatation of the os uteri by the substitution of false pains of a very severe character which give much unnecessary suffering, and exhaust the strength of the patient before the expulsive stage comes on.

The conditions of the bladder which I have found thus to interfere with the first stage of labour are:

1. Complete prolapsus, with or without distension.
2. Partial prolapsus, i.e. where the fundus remains between the uterus and symphysis pubis.

3. Distension while *in situ.*

4. Irritability ?

They all appear to affect the progress of labour in the same way, and the attendant symptoms are similar in each, and very characteristic.

1. *Prolapsus.*—This displacement does not occur suddenly during labour, but in most cases is present before impregnation, as a result usually of repeated child-bearing. It may give rise to little or no inconvenience during pregnancy, and not uncommonly gestation goes to its full term, with no further annoyance than a frequent desire to pass water. Sometimes, however, the urine cannot be retained, and it dribbles away constantly, or the bladder is never thoroughly emptied. At any time during the later months of pregnancy the bladder may become distended, false pains of great severity may be set up, and labour may even be induced prematurely.

More commonly, however, gestation goes on to the full term, with only the slighter inconveniences first mentioned, and only when labour commences does this condition of the bladder give rise to serious trouble. The preliminary uterine contractions, generally painless, and often unnoticed, affect the prolapsed bladder ; there is usually frequent desire to pass water, and if the bladder cannot empty itself the urine accumulates and distension results. But even if the bladder remains empty, the contractions of the uterus are soon accompanied with pain in the bladder, from pressure or traction, which becomes more and more severe. This pain seems to excite the uterus to more frequent contractions, and looking only at the frequency and sharpness of the pains, the labour may appear to be progressing rapidly. The pains, however, have a peculiar and distinctive character. The intervals are shorter than in the early stage of natural labour, and when inquiry is made, the pain is referred not so much to the back, as to the "bottom of the stomach," by which phrase women often mean not the hypogastric region merely, but the vagina and vulva ; in this case its real seat is the bladder.

The appearance and behaviour of the patient is different ; so long as she chooses to remain on her feet and walk about, instead of placing the hands on the loins she prefers to lean on the back of a chair, or to sit down bending forwards with the hands or elbows on her knees, and though these attitudes are common in natural labour, a constant resort to them early in the first stage, generally indicates other than the ordinary pain, and particularly when the pains come on more rapidly than usual. With this there is more frowning, more active contortion of

the features, biting of the lip, and, what is very characteristic, violent straining or forcing; not three or four prolonged efforts, as in the expulsive stage of labour, but in a series of short spasmodic jerks, almost involuntary, attended with great pain and expressions of impatience and suffering. The patient will often say she never had such pains before.

Frequently the patient, misled by the severity of the pains, their "forcing" character, and by the sensation of fullness and distension in the lower part of the vagina, imagines that the labour is near its termination, and betakes herself to bed at an unusually early period.

If the hand is placed on the abdomen, it will be found that the increased firmness of the uterus indicating a commencing contraction is at once followed by spasm of the abdominal muscles, and an examination per vaginam shows the same thing; first tension of the cervix uteri, or of the membranes, if they can be felt, from the action of the uterus, and then the uterus itself is forced downwards into the pelvis by a series of spasmodic contractions of the abdominal muscles. When this takes place, the normal action of the uterus appears to be suspended, as if replaced by the abnormal actions induced, or it operates under great disadvantage. At any rate little dilatation of the os uteri is effected, the labour is greatly protracted, and the patient suffers much unnecessary pain.

There may or may not be symptoms directly calling attention to the bladder, frequent desire to pass water or inability to do so, or both, and there may or may not be accumulation of urine in the bladder. Sometimes also prolapsus, with distension, may exist without giving rise to any special symptoms in the early stages of labour.

The prolapsed condition of the bladder is readily recognized on examination, especially when it contains urine in any considerable quantity. The cavity of the pelvis is found to be occupied by a bag of fluid easily distinguished from the foetal membranes by the fact that it springs from the pubis, and does not permit the finger to pass between it and the symphysis. As this sac, the bladder, fills up the hollow of the sacrum, the os uteri cannot be reached till the urine is evacuated, and if this is done by the catheter, the instrument can be felt from the vagina and followed to every part of the bladder. When the bladder is perfectly empty, the displacement may be overlooked, but the finger, instead of circumscribing readily the lower segment of the uterus, meets anteriorly with the bladder passing from the symphysis pubis to the uterus, and usually disposed in rugae; the introduction of the catheter at

once makes the case clear. The os uteri is almost invariably found high up and far back, so that the anterior part of the lower segment of the uterus, which may be felt in the vagina, and along which is spread the prolapsed bladder, presents an unusually extensive surface. I am disposed, indeed, to believe that the displacement of the bladder is for the most part secondary to the displacement of the uterus.

2. *Partial prolapse* I have considered worthy of mention distinct from complete displacement, because it is more likely to be overlooked, and because the symptoms, though nearly the same, are usually even more severe. There is almost constantly a frequent desire to pass water, and the urine never accumulates. On examination, the lower part of the posterior wall of the bladder is felt in firm small rugae behind the pubis, generally tender when touched, and the seat of extreme pain when the uterus is forced down into the pelvis. The catheter may be felt to pass up between the cervix uteri and the symphysis pubis, showing that the prolapse is not complete. The pains have the spasmodic straining character before described, the uterine contractions affect the displaced bladder, causing pain and irritation; as a consequence violent reflex action of the abdominal and perineal muscles is set up, a kind of tenesmus, which supersedes the normal uterine action, and thus not only is the first stage of labour rendered long and exceedingly painful, but the patient is more exhausted than by a prolonged first stage without this complication.

I have enumerated as other conditions of the bladder affecting the first stage of labour in a similar manner, distension while *in situ*, and "irritability." Cases will be given which, I think, justify me in including them, and I pass them over without further remark than that the symptoms are of the same character, but usually not of the same degree of severity.

Treatment.—The treatment in these cases is simple, and often very effectual. Whenever the symptoms I have mentioned arise, pain in the region of the bladder, with premature involuntary straining, attention should be directed to the bladder, and I think it best always to introduce the catheter. Should any accumulation of urine be detected in the vaginal examination it is absolutely necessary. The patient should also be placed on her back; this not only relieves the bladder from a certain degree of pressure by the falling back of the uterus, but in those cases (and I believe they form a large proportion), in which there is an unnatural position of the fundus of the uterus forwards, and of the cervix backwards, the supine position will aid in bringing the axis of the uterus into correspondence with the axis of the brim of the pelvis. In some cases, also,

much good may be done by drawing the anterior lip of the os uteri downwards and forwards, during an interval, and fixing it during a pain in this position. A third means, to which I should unhesitatingly resort, is the administration of chloroform. The effect is to put a stop to the violent spasmodic action of the abdominal muscles, which is of a sensori-motor character, while the proper uterine contractions, being not interfered with, either by the straining effort, or by the anaesthetic, effect the dilatation of the os uteri. The head once engaged in the pelvis, the displacement of the bladder, though it may still cause great suffering, does not interfere with the progress of the labour, provided, of course, that accumulation of urine has been guarded against.

Influence of Position in Labour.—I may here introduce the few remarks I have to make on the influence of position in labour. The advantages of the supine position in cases of relaxation of the abdominal wall with falling forwards of the uterus, have long been recognized, but they are not confined to instances in which this condition exists. Delay in the going up of the anterior lip of the os uteri perhaps more commonly marks the fact that the descent of the head into the pelvis is slow, than itself operates as a cause of obstruction to this descent. It will always, however, be an advantage to have it out of the way, and this may often be effected by placing the patient on her back. Any one may observe, as I have often done, the difference which this change of position from the side to the back, makes in the position of the anterior lip of the os uteri, and many members of the Society may have seen cases in which it has been attended with a marked change in the rate of progress of the child through the pelvis. One case also I have seen, in which, from the falling of the uterus towards the left side, the head of the child seemed to be directed against the right side of the pelvis, there being further a slight bending in of the ischium on this side ; after long waiting this condition suggested the placing of the patient on her right side, the supine position having been tried with no effect ; the head at once cleared the obstacle, and the labour was speedily terminated, without any apparent increase of force in the pains.

(This paper finishes with the notes of eight illustrative cases.)

CANCER—A NEW METHOD OF TREATMENT

A Paper read at the meeting of the British Medical Association in 1866

British Medical Journal, 1866. Vol. II

The attention of the author was directed to the treatment of cancer under the following circumstances. In 1864, he was consulted by a lady suffering from cancer of the breast. By his advice, the breast was removed by Mr. Walter Coulson. The disease returned, and was again removed in August, 1865. In May of the present year, a tumour was growing more rapidly than ever near the cicatrices of the former operations. It was decided that no further removal was advisable; and, unless something could be done, a miserable fate was before the patient.

The hypodermic syringe is now in the hands of every physician; and it seemed to the author that by it some fluid might be injected into the tumour which might so far alter its structure and modify its nutrition that its growth might be retarded or arrested. After considering the various substances which presented themselves to his notice, he selected acetic acid, for the following reasons. 1. This acid does not coagulate albumen, and might, therefore, be expected to diffuse itself through the tumour, and the effects would not be localized at the point injected. 2. If it entered the circulation, it could do no harm in any way. 3. Acetic acid rapidly dissolves the walls and modifies the nuclei of cells on the microscopic slide, and might be expected to do this when the cells were *in situ*. 4. It had been applied with advantage to common ulcerations.

On May 18 the first injection was practised. The tumour was of about the size of a small egg, and a patch of skin of about the size of a shilling had become adherent to it. The needle was introduced through sound skin an inch or more from the part involved in the disease, and passed to the centre of the mass. About thirty minims of dilute acid (one part of acid to one and a half or two of water) were injected. It gave little or no pain. Next morning, a bulla containing dark bloody fluid was found to occupy the patch of adherent skin. On

May 23, this portion of skin was dry, hard, and horny; the adjacent part of the tumour not so hard. The injection was repeated. The patient was not again seen till June 7, when the piece of skin mentioned was found detached from the surrounding sound skin; and a probe could be passed in all directions to a distance of three-quarters of an inch or more between the tumour and the healthy structures. A little discharge issued from the fissure mentioned. The injection was repeated on this date, and again on the 9th, the acid used being rather stronger. It gave a little pain, and swelling and tension of the parts around followed. On June 13, and a few days afterwards, there was a free discharge of fluid and solid portions, with relief of the swelling, etc. No foetor whatever attended this discharge, which afterwards diminished greatly.

On June 26, on external examination, the tumour was found to be much smaller; and, on passing a probe into the opening, it entered a large cavity extending on all sides. Part of the walls seemed free from malignant structure, but at several points a crust of cancerous deposit remained. On attempting to inject, it was found too thin to retain the fluid, which either entered the tissues and gave great pain, or made its way into the cavity. The cavity was stuffed with lint saturated with dilute acid; and the case left in the care of the family medical attendant, who was to inject as he saw opportunity. July 13. No impression was made on the remaining disease, which had, in the opinion of the medical man, extended somewhat. Carbolic acid was tried for a few days as an application but discontinued; and the cavity dressed daily with strong acetic acid by the medical attendant, and injections practised daily. This energetic treatment gave much pain, and excited inflammation all round. When again seen by the author on August 4, there had been considerable haemorrhage, which had been arrested by free application of tincture of sesquichloride of iron. The result, however, was apparently the entire removal of the remains of malignant disease; and, when last seen, a healthy granulating surface was left at every point. Three others cases were related by the author.

The author further formulated certain conclusions from the experiments detailed, and stated the cases to which, in his opinion, the treatment was not applicable. Guided by his experience, he considered large quantities of dilute acid preferable to stronger acid; and he would not, without great hesitation, attempt the destruction of any tumour which had not involved the skin. His aim had originally been, as stated in the early part of the paper, not necrosis of malignant tumours, but a modification in their

nutrition. The theoretical grounds for this hope were, that cancer owed its malignancy to its cellular (to use a nomenclature now almost antiquated) or foetal structure; and that in acetic acid we had an agent which might be expected to diffuse itself through the tumour and reach the cells, and, having reached them, to effect changes in their structure, and affect them vitally; while it could scarcely do harm. The results he had brought before the profession at the earliest possible moment. The ultimate value of the treatment he left to be decided by a more extended experience.

A CLINICAL LECTURE ON SLEEPLESSNESS

Delivered at the Medical Graduates' College and Polyclinic
The Lancet, 1900. VOL. I

GENTLEMEN,—Sleeplessness is one of the torments of our age and generation, and we are daily called upon to treat it, but I could not well bring cases before you for demonstration. While, however, I have no actual patients to present and shall not even read notes of cases, it is incumbent on me to treat sleeplessness from a clinical point of view. I am fortunately, therefore, under no necessity to discuss the various theories of sleep, nor have I to entertain you with speculations on the neuron or as to the behaviour of dendrons during sleep such as those which now seem to be the inevitable introduction to all questions in which the nervous system is concerned—to attempt to explain, in effect, the *ignotum per ignotius*, which has a perennial fascination.

One word, however, must be said as to difference in the vascular condition of the cortex of the hemisphere in the sleeping and the waking state. According to old experiments the cerebral cortex is in a condition of anaemia during sleep, and it has been assumed that the blood is shut off by contraction of the arterioles, but we have been assured by Dr. Leonard Hill that the vaso-motor regulation of the blood-supply does not extend to the brain, that no nerves are distributed to the cerebral blood-vessels, and that it has been demonstrated by experiment that the blood-pressure within the cranium is entirely controlled by the ebb and flow of the blood in the splanchnic area. On the other hand, my friend Dr. Alexander Morison shows beautiful preparations of arteries from the pia mater in which these vaso-motor nerves are conspicuously visible, and independently of this direct evidence, one was disposed to wonder why these vessels had muscular walls if there were no nerve-supply. It seems to me that the only possible explanation of many of the disturbances of the functions of the brain from peripheral irritation of various kinds is a reflex influence of the cerebral circulation.

In this lecture I propose to limit myself to the consideration of sleeplessness where it constitutes the special complaint on account

of which the patient seeks advice. To speak of loss of sleep where it is only one among many symptoms of disease of brain, lungs, or heart, or one of the effects of fever or other acute affection, would require two or three lectures.

Sleep consists essentially of a suspension of the functions of the higher centres of sensation, or rather perception, involving inaction of the corresponding motor centre. The reflex action of the cord persists as well as the vital reflexes concerned in respiration and the action of the heart. Reflexes also survive in which there is a considerable degree of purposive co-ordination; the extremities will be withdrawn from a source of irritation and we turn over in bed and may adjust bedclothes without waking. The Indian thief will steal the blanket from under the sleeper by so tickling him that he rolls over first in one direction and then in another. While sensation is suspended a loud noise or a bright light, a shake or a sharp impression on the surface, will penetrate the barrier opposed to impressions and rouse the sleeper, and many persons have the faculty of waking at a given hour. Most of us, indeed, can wake up to catch an early morning train in case of need. The intensity of the respective sensory impression required to disturb sleep varies greatly in different individuals and in the same person at different times. A susceptibility to the disturbing influence of light and sound can be cultivated by sleeping in darkened rooms and with closed curtains. On the other hand, habit enables a Londoner to sleep, not only in spite of the more or less continuous roar of traffic, but through the thunder of a railway wagon or a post-office van tearing along a quiet street, and many persons can sleep in broad daylight.

Misuse of Drugs.—It may be said at once that the treatment of sleeplessness does not resolve itself into a choice of drugs. To compel sleep by opiates or sedatives is not to cure sleeplessness. It is true that when the brain has been overtaxed by engrossing work, or the nervous system has been shattered by a severe shock or exhausted by overwhelming anxiety or outworn by excitement, or the habit of sleep has been broken by long and anxious vigils over a sick bed, a judiciously selected remedy may quiet the molecular vibrations and restore self-control to the brain and so break the wakeful habit and renew the patient's confidence. Setting aside these exceptional cases the sedative, whatever it may have been, may leave the cause of sleeplessness untouched, and while this remains in full operation the nervous system becomes accustomed to the drug and a larger and larger dose is required. Then, moreover, the effects of the drug are not confined to the production of sleep. The substance is carried by the blood everywhere, and it may

check secretions, derange digestion, impair the peristaltic vigour of the stomach and intestine, affect the circulation and deteriorate the blood, and interfere with the nutrition of the tissues. One effect all drugs have, and always have—they diminish the resistance and impair the manhood of the individual. It is not only that he has experienced relief and that he longs for it again, but he will not endure with patience and fortitude a privation of sleep which he would formerly have thought unimportant. So it goes on until the morphia habit is established or the patient becomes a slave to chloral or sulphonal or trional.

Nothing is easier than to obtain a cheap kind of credit by prescribing a sedative, especially if its name is new to the patient and he can be assured that he is not taking an opiate properly speaking; but it may be the first step in the downward course towards suffering, bodily and mental, of the most terrible kind. From what I have seen I should prefer to be a victim to morphia or to opium rather than to chloral or to sulphonal or to trional. If the reaction from the opium intoxication is painful there is at any rate a positive pleasurable exaltation, whereas the best that chloral and sulphonal can give is oblivion, and there is not only depression but a pitiable loss of volition. The wretched subject cannot make up his mind on the most trivial question, and when he has come to a decision he regrets it immediately. Besides this indecision there is loss of memory, and either from this loss of memory or from impairment of the moral sense no dependence is to be placed on his statements. The heart and the vessels lose their tone, the circulation becomes languid, and the tissues grow soft and flabby, till finally the victim is incapable alike of effort and enjoyment. Unfortunately these drugs are placed within the reach of all in the form of syrups and tabloids; there is no restriction on their sale, and they are constantly taken on the advice of chemists or friends. The medical man who prescribes any of them has thus no further control over their administration and incurs therefore a terrible responsibility.

Causes. Nervous system.—The essential preliminary to the treatment of sleeplessness is the recognition of its cause in the particular case. We have therefore to consider the causes of sleeplessness. Of these the most important undoubtedly is the original constitution of the nervous system. The capacity for sleep and the readiness to sleep vary enormously in different persons. Some can command sleep at a moment's notice almost at any time and under any circumstance; with others sleep always requires careful wooing. But while there are inborn differences in this respect manifested even in infancy, much can be done to

bring the nervous system into a condition which favours sleep or the reverse, and this is a point always to be borne in mind in the treatment of sleeplessness. Of all the influences which tend to bring the nervous system into a state in which sleep is ready, sound, and refreshing, the most important are fresh air and exercise. A sedentary mode of life has a contrary effect. Besides, therefore protecting the light sleeper from all causes of sleeplessness and removing any which may be identified, it may sometimes be necessary to revolutionize his habits and to lay down special rules as to his mode of life. The old distich "After dinner rest awhile, after supper walk a mile" seems to have been written for the benefit of such an individual.

These naturally bad sleepers are greatly to be pitied. The slightest change in the bed or of pillows or coverings will give a bad night; the pillow must be high and firm for some and it must be soft and low for others. Sleep in a strange room is for a time impossible. A chink in a shutter, or a badly-fitting blind, or ill-adjusted curtain admitting a single beam of light will cause some to wake up at daybreak. Some must have absolute darkness, others require a certain degree of artificial light. It is in such cases that the great difficulty arises of deciding whether sedatives of one kind or another may be given. Much depends on the kind of sleeplessness. If the patient can lie quiet and, while sleepless, remain tranquil, I should hesitate to give drugs. He has rest even if he does not sleep, and very commonly there is sleep of which he is not conscious. An occasional dose may be given to parry the effects of any unusual excitement or fatigue. The choice of the drug would be determined a good deal by the experience of the individual, but if the pulse tension were high chloral would be indicated as a vascular relaxant; if it were low, paraldehyde or bromide or some such combination as morphia and hyoscyamus. When the patient tosses and fidgets and gets up looking worn and tired, there is more need to have recourse to sedatives, and it is extremely likely that their use will become more or less habitual. If possible bromides should be the drugs employed. There are cases in which a moderate dose of ammonium bromide just seems to bring the irritable and sensitive nervous system to an average condition and I have known patients to take it indefinitely without apparent injury. I cannot say this of any of the chloral and sulphonal class of drugs.

Cold Feet.—There are conditions of the circulation which interfere with sleep. Perhaps the most easily recognized of these is coldness of the feet. Anaemic girls are often kept awake by cold feet, and in debility from other causes coldness of the extremities may prevent

sleep. For sleeplessness so caused a hot bottle is the obvious remedy, or, perhaps better, enveloping the legs in warm flannel up to and above the knees, which will usually be felt to be cold as well as the feet. Such patients should be warned not to dawdle while undressing but to get into bed quickly, and the feet should be vigorously rubbed. To anything of this kind, however, they are indisposed by the very languor and weakness which give rise to the coldness of the extremities. A great help to sleep in patients suffering from sluggish circulation is a little very hot and strong beef tea or hot milk on going to bed. Stimulants should be avoided in the case of the young; there is less objection to them in elderly people, but in them the stimulant should not be relied upon simply as such, but should be given in the form of a hot drink, such as negus or spirit and hot water.

Cold feet may prevent sleep when there is no weakness of any kind after hard intellectual work, especially when carried on late into the night. It is not a mere negative coldness which can be rectified by supplying warmth; external heat seems to be rejected and the feet refuse to be warmed by it. There is, in fact, spasm of the arterioles excluding the blood, just as when the feet are stone cold in fever while the temperature in the mouth is 103° F., and it may be pointed out that when the extremities are warmed by the application of external heat the warmth is not simply supplied from without; the vessels are relaxed by the heat applied to the surface and the warm blood is admitted. Under the circumstances we are considering, the reflex vaso-dilatation does not take place, and the hot bottle, even if available, is of no use. The resource is friction, but this has to be persevered with for some time to be effectual. To stand in cold water for a few minutes before rubbing the feet is an excellent expedient, resorted to, as I have found, by many hard-working friends, and to which I can bear personal testimony. Hot beef tea or milk would be invaluable, or even a drink of hot water, but brain-workers are too much engrossed with the task in hand for forethought to provide these remedies.

Hot Feet.—Sleeplessness may be due to an exactly opposite condition—a hot burning feeling in the feet which, however, more commonly wakes the patient up than prevents his getting to sleep. It may be part of a general sense of heat and discomfort attending gout, or subacute rheumatism, or rheumatoid arthritis, when the feet may be really hot, or the sensation of heat may be subjective only, while the feet are cold to the touch and deep crimson in colour. Patients will often insist on putting the feet out of bed, but the relief thus obtained is only partial and temporary. The treatment

of the sleeplessness will be that of the underlying condition. If an opiate is required, as may be the case, especially when there is pain, an old-fashioned Dover's powder is one of the best vehicles, and there is little danger of its laying the foundation of the opium habit. Or phenacetin or antipyrin may afford relief and find a legitimate opportunity of usefulness.

High Arterial Tension.—Conditions of the circulation which do not give rise to such conspicuous effects are very frequently attended with sleeplessness. One of these is high arterial tension. It may be supposed that the high blood-pressure does not permit the arterioles to shut off the supply and reduce the cortical circulation. Whatever the explanation may be the cases are very common in which high arterial tension and sleeplessness go together and in which the lowering of the one affords relief to the other. A characteristic of this form of sleeplessness is the impossibility of getting off to sleep.

Since the presence in the blood of nitrogenized waste is one cause of high tension the treatment dictated is to minimize its formation and promote its elimination. The patient will be put on a regulated diet in which the proportion of animal food will be adjusted to the individual requirements. In this case the patient's previous habits and dietetic idiosyncrasies must be taken into account. Nothing can be more absurd than to lay down rules to be applied indiscriminately to all sorts of constitutions. A glass of water night and morning is usually a good thing. Among the most efficacious of the eliminants is a mercurial aperient which, as it may have to be repeated once or twice a week for a long time, must be mild. A single grain of calomel or one or two of pil. hydrarg., or hyd. cum cret. with colocynth and hyoscyamus, or compound rhubarb pill, is usually sufficient, and may be taken twice a week indefinitely. The eliminant action of the mercurial aperient may be followed up by mild salines or by alkaline tonics. Chloral has a specially favourable hypnotic influence when the blood pressure is high, as it relaxes the peripheral vessels, and it owes much of its credit to its good effects in these cases.

Low Arterial Tension.—Rare instances are met with in which an exactly opposite condition—extremely low tension—appears to give rise to sleeplessness, or at any rate makes sleep in the horizontal position difficult or impossible. In the sitting position these patients can scarcely keep awake; at any period of the day they will drop off to sleep if they sit down and try to read, and even over the morning newspaper; and after dinner they will sleep indefinitely until they go to bed and lie down when at once they are wide awake. It seems as if the toneless vessels were incompetent

to resist the slight increase of pressure within the cerebral vessels when the patient lies down, the cortical capillaries being full when the head is low, empty when it is raised. A similar effect is seen in some forms of heart disease, but produced by venous obstruction rather than by arterial asthenia. Sleep is impossible in the recumbent position, and the worn-out sufferer spends the weary night upright in his chair.

The remedy in low tension sleeplessness is not a narcotic, but a cardio-vascular tonic. Digitalis, or theobromine, or caffein will be given as well as the particular tonic indicated by the general condition. But very frequently a tea-cup of strong hot beef tea will send the patient to sleep at once, or even a cup of tea or coffee, which are generally antagonistic to sleep. Beef tea is better than milk, as imposing less work on the stomach, and the temperature is a matter of importance; the quantity must be small.

Low pulse tension is probably a factor in the sleeplessness of acute febrile diseases, though the main cause is a toxin. The effect of a cold bath, or of tepid or cold sponging in inducing sleep in pyrexia, of whatever kind, must be due to the tonic influence on the cardio-vascular system, and is attended with improvement in the pulse tension. A patient suffering from delirium tremens, with its persistent and obstinate wakefulness, may be at once sent into a sound sleep by vigorous cold affusion, even when full doses of hypodermic morphia or hyoscine seem to have no effect.

Flatulent Distension of the Stomach.—By far the most common cause of sleeplessness is indigestion in its various forms, and the particular incident of indigestion which seems to be the most active opponent of sleep is flatulence, especially gaseous distension of the stomach. Apparently it is mechanical pressure or stretching which prevents sleep, since sleep often at once follows the eructation of a few cubic inches of gas. The volume of gas displaced is quite insufficient to affect the splanchnic circulation, and it is not easy to understand in what direction the pressure operates which produces the result. There may be extreme dilatation of the stomach from pyloric obstruction without serious influence on sleep, and whenever the stomach has been displaced downwards, so that the lesser curvature is defined on the abdominal wall, or can be followed by palpation, I have found that the effect on sleep was little marked. When, on the other hand, the upper line of gastric resonance has been high, corresponding with the fifth space and the base of the ensiform, sleeplessness is common even though the area of resonance may not be very extensive.

It does not follow that when flatulence is the cause of sleeplessness the patient should suffer from the ordinary

symptoms of flatulent dyspepsia, epigastric pain or discomfort, and frequent eructations. Flatulent distension of the stomach produces its worst effects when we are not conscious of its existence. When we suffer from flatulence in the ordinary sense of the word, what we are conscious of is not the flatulence itself but the effort to get rid of or displace it. When it is tolerated by the stomach and no effort is made to expel it, its more serious effects are produced—anginoid pain in the cardiac region, palpitation of the heart, and sleeplessness.

It will be clear from what has been already said that the form of dyspepsia attended with sleeplessness is that in which the special characteristic is atony of the muscular walls of the stomach, allowing of the passive distension of the organ. A further inference will be obvious—that it is in brain-workers whose nervous energy is diverted from the work of digestion, or in those whose mode of life is sedentary and whose general nerve-tone is low, or in persons depressed by anxiety and worry, that this form of dyspepsia is most liable to occur. One way, indeed, in which grief and worry and anxiety give rise to protracted sleeplessness is through the effects on digestion. Sleep will come to the relief of grief, and even anxiety gives way to weariness and sleep, but nothing is more certain than the effect of mental depression on digestion, and, when the sufferer is roused by flatulence or abdominal discomfort, the distressing or agitating idea takes possession of the mind and banishes further sleep. Flatulent dyspepsia, again, is one of the causes of the sleeplessness which is said to belong to old age. It is not old age as such to which sleeplessness is to be attributed but to the infirmities attending it—to vascular conditions or functional derangements of one kind or another of stomach, bowel, or bladder.

Flatulent dyspepsia very often interferes with sleep on first going to bed and may keep the sufferer awake indefinitely. This faculty it shares with various causes of sleeplessness, but one very common variety of sleeplessness is highly characteristic of flatulence. The patient, possibly a good sleeper naturally, falls asleep on getting into bed and then wakes punctually night after night at a given hour, at two o'clock, three o'clock, or four o'clock as the case may be, and then lies awake for the rest of the night. The explanation is that the last meal has not been entirely passed on into the duodenum. Fermentation takes place in the food which remains in the stomach, and after a certain time sufficient gas and acidity have been developed to disturb the sleep.

The treatment of sleeplessness due to flatulence is, of course, that of the dyspepsia. This in itself would demand an entire lecture for its discussion and you will not expect me to enter upon

it. All I need do is to enumerate the expedients for preventing the interference with sleep. The simplest of these is a tumbler of hot water at bedtime, and it is usually effectual. The stomach is stimulated to contract, much of the gas present in it at the time is expelled, and any fermenting contents are swept on into the small intestine, where the antiseptic bile checks further fermentation and where flatulence gives rise to less disturbance. The hot water should be taken before undressing, so that it may have time to clear out the stomach and expel the gas before the patient lies down. Should hot water not be sufficient sal volatile and carbonate of soda may be taken before it or an alkaline carminative draught may be given—carbonate and sulpho-carbolate of soda with aromatic spirit of ammonia, compound tincture of chloroform, or ether and peppermint or camphor water, and sometimes bromide of sodium or ammonium may be added with advantage for a time. Friction over the epigastrium or between the shoulders may help to disperse the flatulence. I do not think it is well to allow even so simple and harmless a matter as a nightly dose of hot water to grow into a habit. As a rule it should not be continued for more than a week at a time as the response of the stomach becomes imperfect. It has seemed to me that an alkaline draught can be taken longer without losing its effect. I know several patients who wash out their own stomach at bedtime whenever they have sensations which lead them to apprehend a bad night.

Some individual causes of sleeplessness must be considered. *Tea* and *coffee* are looked upon as powerful agents in the prevention of sleep, and there are no doubt many here who have found a cup of strong tea or coffee an excellent preparation for an evening's work. Certain kinds of tea and really good coffee do undoubtedly act as stimulants to the brain ; they help the tired nurse to keep awake and fresh, and keep sleep at bay for the student who is working far into the night. They may, indeed, produce a condition of intense wakefulness. When, however, it is stated that a cup of tea in the afternoon will keep a given person awake all night, I doubt very much whether it is the tea, as such, which is responsible for the result. Imagination often plays a very influential part in the effect. Let such a person be confidently assured that pure China tea, brewed for not more than two minutes, is innocent of such evil properties and he or she will often take it with impunity whether it conforms exactly to the description or not. But afternoon tea is a very common cause of flatulent dyspepsia and in this way may be responsible as the cause of protracted sleeplessness. So with the after-dinner cup of black coffee, it is often the dinner and not the coffee which disturbs the night's rest.

Influenza.—Influenza has familiarized us with sleeplessness of a most obstinate character. There is always asthenia, cardio-vascular and nervous, which must be borne in mind in the treatment. With the general effect on the nervous system there may be complications which contribute seriously to the prevention of sleep and may even interfere with the action of powerful opiates or sedatives. Among these is acute dilatation of the stomach, a not uncommon incident of an attack of influenza. It was present in the worst case of post-influenzal sleeplessness that I ever saw, in which there was not a wink of sleep for four days and nights. Sleeplessness following influenza must be treated as an acute affection and, unless there is speedy improvement under such tonics as arsenic or phosphorus, strychnine and quinine, together with measures for the relief of functional derangements, opiates may be given without hesitation, and I think it better to have recourse at once to combinations of opium or morphine and hyoscyamus with carminatives than to try sulphonal, or trional, or chloral and bromides. In case of need morphine may be given hypodermically, and it usually adds greatly to the efficiency to combine it with strychnine as well as atropine.

The immediate effect of large doses of alcohol is torpor. A result of long-continued alcoholic excess is sleeplessness culminating in delirium tremens. The remedy here is total abstinence with considerable doses of strychnine or nux vomica and perhaps digitalis. At the same time the liver and stomach disorders resulting from the alcohol will demand attention, as they may keep up the sleeplessness and are indeed frequently its main cause.

I do not know whether it is worth while mentioning some of the popular remedies for sleeplessness. Among them is the hop pillow which certainly sometimes seems to soothe. There is, again, the saffron bag applied to the pit of the stomach, immortalized by Bulwer Lytton in *The Caxtons*. Gently smoothing the hair is undoubtedly efficacious in many cases, as is also, but less frequently, sponging the burning palms. Dipping the face in cold water is one of the expedients of the worker late at night; some will sponge the entire head. Then there are the different ways in which people try to hypnotize themselves by watching and counting the invisible breath, slowly counting imaginary sheep as they pass through an imaginary gate, and the like. I have not ventured on the large subject of the employment of hypnotism. That it has its legitimate uses in inducing sleep I have no doubt. It has not been my good fortune to meet with a case where it has overcome the morphine habit or rescued the victim of chloral or sulphonal.

WARBURG'S TINCTURE

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WARBURG'S tincture has long held a high reputation in India, as a remedy of undoubted and indeed unequalled power in the treatment of the malignant malarial fevers of that country and of cholera. Testimony to its efficacy has come from men whose capability and opportunities of forming an opinion could not be disputed, but until quite lately it was a secret remedy, and in this country we are not frequently brought face to face with the terrible emergencies in which its efficacy has been shown, and have not, therefore, been compelled to sacrifice our objections against the employment of an unknown agent to the safety and welfare of the patient.

My interest in Warburg's tincture had been excited by published statements and private testimony as to its value; when, therefore, Professor McLean made known its composition and mode of preparation in the *Medical Times and Gazette* for 1875, Vol. II. p. 540, giving his unqualified support to all that had been said in its favour, I made a careful study of it, with a view to ascertain the therapeutic principles on which its effects depended.

The following is the formula made known by Professor McLean :—

Aloes (Socotr.) libram;
Rad. Rhei. (East India);
Sem. Angelicae.
a. Confect. Damocratis; ana uncias quatuor.
Rad. Helenii (s. Enulae);
Croci Sativi;
Sem. Foenicul.
b. Cret. Praeparat; ana uncias duas.
Rad. Gentianae;
Rad. Zedoariae;
Pip. Cubeb;
Myrrh elect.;
Camphor.
c. Bolet. Laricis; ana unciam.

The above ingredients are to be digested with 500 oz. proof spirit in a water-bath for twelve hours; then expressed and 10 oz. of disulphate of quinine added; the mixture to be replaced into the water-bath till all the quinine be dissolved. The liquor when cool is to be filtered, and is then fit for use.

a. This confection, which consists of an immense variety of aromatic

substances, was once officinal, and is to be found in the London Pharmacopoeia, 1746.

b. Dr. Warburg states that this ingredient was added to correct the otherwise extremely acrid taste of the tincture. Many other substances were tried, but none answered so well as prepared chalk.

c. This is the Polyporus laricis (P. officinalis, Boletus purgans, or larch-agaric), "formerly," says Pereira, "used as a drastic purgative, and still kept by the herbalist."

The tincture is of a deep brown colour, has an aromatic and slightly terebinthinate odour, and an intensely bitter and warm aromatic taste; a noteworthy point about it is that there is nothing spirituous in either taste or smell, and it seems as if the alcohol employed in its manufacture were entirely saturated, and so to speak extinguished, by the substances taken up. The tincture evaporates readily, leaving a copious brown residue.

"It will be seen," to employ Professor McLean's words, "that quinine is the most important ingredient in the formula, each ounce bottle containing nine grains and a half of the alkaloid. Its presence has been detected by every chemist who has attempted its analysis, and never doubted by any medical man of experience who has used the tincture. Many will say," continues Professor McLean—"'After all, this vaunted remedy is only quinine concealed in a farrago of inert substances for purposes of mystification.' To this objection my answer is—I have treated remittent fevers of every degree of severity, contracted in the jungles of the Deccan and Mysore, at the base of mountain ranges in India, on the Coromandel Coast, in the pestilential highlands of the northern division of the Madras Presidency, on the malarial rivers of China, and in men brought to Netley Hospital from the swamps of the Gold Coast, and I affirm that I have never seen quinine, when given alone, act in the manner characteristic of this tincture. And although I yield to no one in my high opinion of the inestimable value of quinine, I have never seen a single dose of it given alone, to the extent of nine grains and a half, suffice to arrest an exacerbation of remittent fever, much less prevent its recurrence, while nothing is more common than to see the same quantity of the alkaloid in Warburg's tincture bring about similar results."

I will give two illustrations of the effect of Warburg's tincture which have come under my observation.

The first is that of a gentleman, seen with me by Dr. Clarke, of Huddersfield, whose medical history is briefly as follows:—His father suffered much from gout, but lived to the age of 84 or 85. He had himself had little illness of any kind. Early in 1876 he took cold and had bronchitis, and together with this a febrile condition, attended with severe pains in the limbs, which were worse at night. He was unable to sleep, had no appetite,

vomited his food, suffered from constipation, and in consequence rapidly lost strength. At length, while taking small doses of iodide of potassium with ammonia, colchicum, and bark, given on the hypothesis that the pain was of gouty origin, he began to improve. On his eightieth birthday, which was in the second week of March, he was convalescing slowly, and was sitting up in his room for the second or third time ; he was wishful to show, by walking without assistance, that he was not so helpless as he was made out to be by his family ; he had miscalculated his strength, however, and overbalancing himself, fell backwards, striking his hip against the fender. The pain in the injured hip was so great that complete examination was impracticable, and it was supposed for some time that there was intra-capsular fracture of the neck of the femur.

The shock of this accident brought back the old symptoms, pain, sleeplessness, restlessness, inability to take food, constipation, and vomiting. The patient was rendered so helpless by the pain in the hip, caused by the injury, and the pains in the limbs, together with weakness, that he was unable to turn in bed, and the lower extremities were for a time absolutely powerless, and had to be shifted for him. He rallied from the immediate effects of the fall, and having shown the constitutional vigour which this implied, it was hoped he would continue to improve. This, however, was not the case, the helplessness continued, and, worn out by pain, sleeplessness, and inability to take food, the strength rapidly failed, the desire for, and expectation of, recovery, which had hitherto been a great source of hope, ceased, and for the first time the mental faculties became impaired, and there were forgetfulness, wandering, hallucinations, etc. ; the pulse was frequent and weak, and the tongue became dry. It was observed that there were febrile accessions, attended with flushing of the face, but not giving rise to any notable elevation of temperature, and the state of the case appeared to be that while the heart and vessels were wonderfully sound, the kidneys healthy, the nervous system vigorous, the strength was being worn out by suffering, sleeplessness, and fever.

This was exactly the condition in which, as it seemed to us, Warburg's tincture might find its application, and, everything else having failed, it was resolved to make trial of it. This was Sunday, April 9, but it was not till the following Thursday that the tincture could be procured, by which time it appeared too late for any remedial measures. During Thursday night the patient was wildly but feebly delirious ; he had ceased to recognize the members of his family ; there were general muscular tremor

and jactitations, and all the signs of approaching death. Two doses of the tincture were given on Friday morning, but without any hope of good result, and I was summoned by telegraph. On my arrival in the afternoon, instead of finding the patient moribund or dead, as I feared, he was in a quiet sleep and bathed in a warm perspiration, while the whole room was fragrant with the aromatic constituents exhaled by the lungs and skin. The sleep continued for twenty-four hours, interrupted only by the administration of food, and he woke from it refreshed, free from pain, and quite clear in mind, though, of course, extremely weak. From this time there has been slow but continuous improvement, and the patient now eats and sleeps well, is free from pain, cheerful, and in the full enjoyment of his intellectual faculties, though he remains very helpless as regards muscular power, and appears indeed to have had spinal meningitis, as there is some degree of wasting and contraction of the limbs. One good effect of the tincture, which he has taken in small doses ever since, has been its mild but efficient aperient action on the bowels.

A second case, in which it appeared to me that Warburg's tincture certainly saved life, was that of a boy of 6, in whom scarlet fever was accompanied by extreme frequency of pulse, and especially of respiration. A favourable opinion as to his chances of recovery was entertained, notwithstanding a pulse of 160 to 180, and respirations 60 per minute, chiefly because his aspect was good and the mind fairly clear, the beats of the pulse also remaining distinct, till the morning of the seventh day, when the pulse was not only more frequent than ever, but the beats ran into each other, and the boy was scarcely conscious. On the evening of this day the aspect of the boy was still worse, and indeed hopeless. Warburg was now given ; the first dose was vomited, but a second was retained. Next morning, though he had a death-like pallor and the pulse was scarcely perceptible, the beats were distinct. The stomach rejected everything, and the Warburg was given by the rectum with beef tea and brandy, and in spite of complications the boy recovered.

My chief purpose in this communication, however, is not to extol Warburg's tincture. Nothing, indeed, that I could say would add weight to the testimony of Professor McLean. I have sought, as is my habit, by study of an undoubted therapeutical fact to arrive at the principles on which the effects are produced, and if possible to ascertain the mode of action of the remedy.

As will have been seen at once, even without the remarks quoted from Professor McLean, the main ingredient is quinine, and one great principle of its action is the *combination of quinine with power-*

ful aromatics. It is aperient also from the aloes, rhubarb, etc., contained in it, and this may contribute to its usefulness, though in a minor degree only. It is a fact with which physicians are perfectly familiar, that the action of quinine is often aided by stimulant and aromatic adjuvants—the compound tincture of bark is testimony to this; perhaps the advantages of the combination have not been borne in mind in the same degree when large doses of quinine have been given for the purpose of reducing pyrexia or restoring power in adynamic conditions, as some forms of pneumonia.

A large dose of quinine sometimes not only gives rise to the usual symptoms of cinchonism, but produces great prostration, and I have distinctly seen harm result. It appears to me probable that this can be counteracted by aromatic stimulants, and after the experience with Warburg which I have just related, I tried, in a case of extreme prostration at the end of a relapse of enteric fever, large doses of the liquid extract of cinchona and of ether combined. The boy could scarcely have been more pale had he been dead; the temperature was only slightly raised; he was pulseless and unconscious, and did not rally at all when stimulants were given. He began to improve as soon as the bark and ether were given, and eventually recovered. I think a large dose of quinine alone would have killed the boy, and that the ether alone would have failed to raise him.

A combination I have long employed in giving large doses of quinine is with very strong black coffee and brandy.

But there is a second therapeutical principle, of which advantage is taken in the employment of Warburg's tincture—this is *concentration.* The maxim is as old as Hippocrates: "Ad extremos morbos, extrema remedia exquisite optima" (Aphorism vi.). We hear much of the physiological administration of drugs, by which is usually meant the exhibition of small and frequent doses, though no doubt it was really intended by the framers of the phrase to indicate the giving of a remedy in such doses and at such intervals as are indicated by its energy of action, rate of elimination, etc. But there is a *therapeutical administration* of drugs, to which physiological experiment affords no clue, and for which the condition of the patient is the indication.

Concentration in the employment of Drugs.—In *post-partum* haemorrhage a drachm of laudanum will rally the exhausted strength, restore consciousness, rouse the uterus to action, and arrest the bleeding when six times ten minims in water would allow the patient to bleed to death. In syncope a tablespoonful of undiluted brandy will have an effect which twice the quantity drowned in water would fail to produce. We defeat a paroxysm

of ague and cut short the disease, or suppress neuralgia, by a single large dose of quinine, when repeated doses of average amount have entirely failed.

The instructions given with Warburg are that the bowels shall have been freely open, that no food shall have been recently taken, and that two doses shall be administered without dilution at an interval of two or three hours, nothing being taken between but a little brandy or beef tea, and this only if required by the state of the patient. The remedy thus enters the empty stomach in a highly concentrated state, and is no doubt rapidly absorbed. Its remarkable effects are distinctly due to the powerful impression made upon the nervous system.

I may call to mind other examples of striking curative effects due to an impression on the nervous system, and it is worthy of remark, how little specific this impression may be, provided only that it is energetic. It is a well-known fact, that strong spirit of camphor, taken on a piece of sugar, will often arrest severe and dangerous diarrhoea, especially in children, sometimes, indeed, when other remedies have failed. Cajuput oil, and probably other essential oils, will sometimes have the same effect. Now what is the condition of success, and what the order of phenomena observed? There is a sense of heat, almost of burning, in the mouth, throat, and stomach; to this succeeds excitement of the pulse, a glow of warmth throughout the system and over the surface, flushing of the face, and often perspiration. Let these effects be produced almost by whatever means, and the desired result usually follows. I have been assured by competent observers that the premonitory diarrhoea of cholera, and even the declared disease at the outset, can be cut short by a combination of strong stimulants, such as ammonia, ether, spirit of lavender, and tincture of capsicum. "You must bring tears into the eyes," said one of my informants.

I have already mentioned the employment of quinine in large doses in intermittent and remittent fevers. Its remarkable influence on the temperature is another illustration of what I have spoken of as the therapeutical administration of a remedy. A single dose of 20 grains will often bring down the temperature in enteric fever from 105° to 100°, or lower, and keep it thereabouts sometimes for twenty-four or thirty-six hours, whereas the same amount distributed over the twenty-four hours would have little appreciable influence.

The effect of salicylic acid again in acute rheumatism appears to depend greatly on the way in which it is employed. I have yet to see the case of genuine acute rheumatism without complication in which the pain is not entirely gone, and the temperature

normal, after six consecutive doses of 20 grains, at intervals of an hour, on two successive days.

I may perhaps be permitted to mention a more modest application of the therapeutic principle of following up an effect, so as to make a decided impression, not for a moment comparable to those just described, but useful in its place. A well-known treatment of catarrh, or of the early stage of bronchitis, is to give acetate or citrate of ammonia with perhaps free ammonia, vinum ipecacuanhae, spirit of nitrous ether or of chloroform, and some preparation of opium. One can only give a certain amount of such a mixture at a dose, but by giving a series of doses, say at 8, 9, and 10 p.m., instead of every three or four hours, we may often cut short an attack which would otherwise go on for days. I have occasionally seen striking instances of prompt relief, especially in the case of patients in whom catarrh was accompanied by asthmatic dyspnoea, tincture of belladonna being then added.

Note by the Editor.

During the attack of pneumonia by which Sir William Broadbent was struck down in October, 1906, he was on the fifth day in a condition of extreme danger, being unconscious with respirations 50 to the minute and an irregular pulse of over 160 with tumultuous action of the heart. Having seen him use Warburg's tincture in similar emergencies, we gave him a rectal injection of half an ounce of the tincture. In about half an hour's time he broke out into a profuse perspiration, and the pulse-rate began to diminish; an hour and a half after the injection the pulse-rate had fallen to 120 and the heart was quite regular, respirations were 40 to the minute and consciousness had returned. Rectal injections of half an ounce of the tincture without aloes were given every four hours for the next two days, till all immediate danger had passed, though the temperature was still above normal.

TWO CASES ILLUSTRATING THE SUCCESSFUL EMPLOYMENT OF THE COLD DOUCHE

Medical Society's Proceedings, March 5, 1883

CASE 1.—*Delirium Tremens.*—The patient was a gentleman, aged about 38. His constitution was thoroughly broken by excesses, especially alcoholic, and he had had several previous attacks of delirium tremens. He had been under my care in one of these in November, 1876, which had been ushered in by severe haemoptysis followed by violent convulsions, and the case had been remarkable from the fact that the characteristic delirium and tremor returned several times after a long sleep and complete recovery of the mental faculties. The first treatment was by digitalis and bromides, no alcohol being allowed, but beef tea being given freely. In four days there was sound sleep, out of which the patient awoke apparently well.

The symptoms, however, gradually returned, and on the third day he was as bad as ever. The bromides and digitalis were again given, brandy being now added to the beef tea, on the supposition that the relapse might have been due to absence of stimulants when the patient's strength was reduced by loss of blood. There was little apparent improvement, and chloral was given with the bromide, when at length sleep was procured. A relapse followed even more promptly than before, upon which morphia was administered subcutaneously, in doses of half a grain. Again sleep was obtained, with apparent recovery, but all the symptoms returned, and twice more was this recovery under morphia and subsequent relapse repeated. I now took advantage of the first moment when such a proceeding could be considered safe, and took the patient out with me. I made him name all the streets we passed through, kept his attention occupied, and when I had to make a visit told the coachman to keep moving, so that he could not leave the carriage. He always had a story to tell when I rejoined him of two women who had been with him "fighting like the devil," and in his imagination we ran over and killed scores of children. However, he slept after the drive, and afterwards he was sent out daily with the nurse in a carriage till he was quite well.

I was called to him again on December 6, 1878, and found him suffering from severe delirium tremens, which had been carried on

for some days. He was well plied with beef tea, had digitalis and bromides in full doses, and, finally, with great reluctance, morphia in grain doses subcutaneously, without effect. On the night of the 9th he was in a condition of extreme exhaustion, the face pale and haggard, the eyes wild, the skin bathed in perspiration, the pulse soft and small, frequent and irregular. He was almost too feeble to turn his head to follow the images of his fantasy, but was constantly muttering and exclaiming, while his fingers fidgeted with the bedclothes, and every limb, or almost every muscle, was the seat of jactitations. Dr. W. A. Smith, now of Newport, Essex, remained with him all night, and on the morning of the 10th reported that there had been no sleep and no cessation of the mutterings and jerkings, and that two or three times he thought the patient was dying, the pulse having become imperceptible, the countenance livid, and the voice almost inaudible.

It was obvious that the nervous system would not respond to drugs of any kind, and that unless it could be roused by some means or other the patient had not many hours to live. I resolved, therefore, to try the douche. Ice-cold water was brought, and a large bath sponge, the patient was stripped to the waist, arrangements were made to protect the bedclothes, and then the sponge, as full of water as possible, was violently dashed against the head, face, neck, and chest. This was done two or three times, the skin being quickly and roughly rubbed dry between with a coarse towel. I will not attempt to describe the gasping and sputtering and impotent swearing. The process was repeated on the back, and the patient then being made comfortable was told to close his eyes and to go to sleep, my hand being firmly placed upon his. This he did at once, but in about five minutes he awoke, apparently disturbed by the jactitations, which continued to be violent. He was obviously better, the pulse more full, firm, and regular, while the face had warmth and colour. During the few minutes of sleep the perspiration could be seen to form drops on his forehead, and roll off almost in a stream.

He could not be made to go off to sleep again, and as his agitation increased, and he employed his renewed strength in struggling and shouting, the douche was again administered as freely as before, and he was afterwards commanded to keep his eyes shut, and go to sleep, as before. He obeyed, sleep came almost instantaneously, and, in spite of the jerking of the muscles, lasted three hours. On waking up this time he asked where the doctor had got that water from, and was there any more like it. If there was he would have it used again. Used it was by Dr. Smith energetically, after which the patient slept continuously for twenty-four hours, only waking to take food. Convalescence followed quickly and satisfactorily.

CASE 2.—*Sleeplessness and Pyrexia after Childbirth.*—I was called on the evening of June 3, 1881, to see a young married lady who on May 30 had been confined of her first child. The labour had been prolonged and severe, the perineum had been ruptured, and the bladder paralysed. From the setting in of labour, and it was said from a day or two before, there had been no sleep whatever. I was detained and did not reach the patient's house till 11.30 p.m. She was under the care of Mr. Ord of Streatham Hill, whom I met in consultation, and who gave me the above account of the case.

Besides the sleeplessness there were pyrexia and severe abdominal pain with great tenderness in the left iliac fossa. The temperature on the previous evening had been 104·5°, on the morning of the day on which I saw her 104°, and at the time of my visit it stood at 104·2°. The patient complained of severe pain in the head, was restless and tossing herself about in bed, her face flushed, the eyes bright, the expression wild and anxious, the skin perspiring everywhere, the pulse 120, and the milk suppressed. On examining the abdomen it was found to be full and large, but not tense, and the respiratory movements of its walls were not arrested. There was great tenderness over the left iliac fossa, but it was complained of on slight contact and superficial pressure, and not much increased by deep pressure. Opium had been given in various forms, and bromides, and, as need scarcely be said, all the precautions against septic infection of the perineal wound had been taken.

In deciding to recommend the employment of the cold douche I concluded that the abdominal respiratory movement excluded peritonitis, while the character of the pyrexia was not that of puerperal fever or of septicaemia; the local tenderness, again, in the left iliac fossa, was not accompanied by any tumefaction suggestive of pelvic cellulitis or ovaritis, and, as has been already said, it was remarkably superficial. It seemed, therefore, that the pyrexia and the sleeplessness were what we had to deal with, and that if they could be overcome there was every reason to expect that the patient would do well, while it was obvious, on the other hand, that persistence of a temperature of 104°, with entire absence of sleep, was attended with grave peril. It was agreed that she should be sponged all over with tepid vinegar and water, and that to the head, chest, and back the cold douche should be applied in the way described in the previous case. These measures were carried out and, as I was informed by Mr. Ord, the patient speedily fell into a calm sleep, the pain in the iliac fossa subsided, and the temperature fell. There was no further complication, and satisfactory convalescence followed.

MANGANESE, NICKEL, AND ZINC IN ANAEMIA, CHLOROSIS, AND ASSOCIATED DISORDERS

Clinical Society's Transactions, 1869

The experiments (to be briefly related in this communication) were undertaken primarily to test an hypothesis arrived at deductively. The starting point was found in the two postulates : 1. That there must be some relation between the substance administered and the organism, on which the effects produced depend. 2. That so far as the substance is concerned, the basis of this relation can only be its chemical properties, using this term in its widest sense. The conclusion arising out of these which constituted the hypothesis to be examined was, "That substances closely allied chemically must have an analogous action on the system, or the diversity in their operation should be capable of explanation on chemical principles." In other words, "chemical groups ought to form therapeutical groups."

No fact in therapeutics is more certain than that iron cures anaemia and chlorosis, and this metal stands at the centre of a group closely allied in chemical properties, which have to it certain well-defined relations. This group, then, furnished the conditions requisite for experiments which might support or overthrow the hypothesis.

A second object also offered itself, which a few remarks will explain. The usual interpretation of the good effects of iron in anaemia is, that it supplies a natural constituent of the blood which is deficient. This, however, if a true explanation at all (which is strenuously denied by some eminent men on grounds which need not be enumerated here), carries us back but a very short step towards a real comprehension of the mode of action of iron. To attain this the question must be answered why iron is a normal constituent of the blood. The answer is not given by simply enumerating the uses which it serves. As understood by me, iron is normally present in the blood, because of the chemical affinity between it and the organic matter of the blood-corpuscles, and it is useful in virtue of the influence which this affinity exerts on the organic processes. According to this view, then, iron does not cure anaemia,

because it is a constituent of healthy blood, i.e. the two do not stand in the relation of cause and effect; but the cure of anaemia and the presence of iron in the blood are alike consequences of the affinity of iron for organic matter, and of the influence of this affinity on organic operations. (The grounds for this opinion cannot be stated here. It is enunciated somewhat dogmatically for the sake of clearness and brevity, and because it is necessary in order to explain the second and practical object of the investigation.)

If the mode of action of iron be that indicated, then an allied metal, having similar relations with the organic proximate principles, will have a similar effect on the organic processes; and it may be that under certain circumstances its curative influence may be even superior to that of iron. This, however, is inherently improbable; but it is not at all unlikely that by the administration together with iron of one or other of the allied metals, the action of the iron may be aided. The object, then, was to ascertain, if possible, the indications for the employment of a particular member of the group as an accessory to iron in any class of cases. Manganese and nickel stand one on one side of iron, the other on the other, as to their general chemical relations; and it seemed worthy of attention, should it be found that they exerted any favourable influence at all, to endeavour to determine what special set of symptoms associated with anaemia indicated the addition of one or other of them to iron.

Manganese.

The first point to be determined was, whether the metals of the iron group could remove anaemia. For this purpose well-marked cases of anaemia were selected, and the chloride of the particular metal substituted for chloride of iron in the mistura ferri c. quassia of the hospital pharmacopoeia, i.e. it was given with a few drops of dilute muriatic acid in infusion of quassia.

Case 1.—*Marked Anaemia with Amenorrhoea.*—Emma B., aet. 18, servant, came as out-patient to St. Mary's Hospital on October, 14, 1867. Her place was rather hard and harassing. She had been ailing for some time; the catamenia had gradually diminished, and had been absent since August. She complained of great weakness, pain in the side and loins, and shortness of breath. The bowels were regular, her appetite very poor. No leucorrhoea. Her aspect was that of marked anaemia. She took two grains of chloride of manganese, with a grain of quinine, three times a day.

November 4 (three weeks). Looks and feels better. November 21. Still better; catamenia had appeared on the 19th. December 9. Ceased to attend, having continued to improve in appearance and strength. The early return of the catamenial flow is worthy of note. The fact that quinine was given diminishes the value of the case.

CASE 2.—*Marked Anaemia with Menorrhagia.*—Caroline W., aet. 19, servant, came under my care at St. Mary's, on November 21, 1867. Ailing ten months. Symptoms—great weariness; palpitation of heart and breathlessness on any exertion; pain in head. Catamenia coming on every seven or fourteen days, and excessive in quantity. Bowels regular, appetite gone. Anaemia most marked; venous murmur extremely loud.

Chloride of manganese given with dilute sulphuric acid instead of hydrochloric on account of the haemorrhage, dose gradually increased to 4 grains.

November 25. Weaker, faints at times, slightly delirious at night. November 28. Better decidedly. She continued to improve with slight fluctuations, and her colour gradually returned. December 5. Aspect better; still anaemic. December 12. More colour. It was now ascertained that she was living very poorly (this she had refused to acknowledge before), and dinners were ordered for her from the St. Mary's kitchen. With this assistance the improvement was still more rapid. On December 30 she felt so much better that she began to talk of taking a place again. On this account I considered it due to her to add iron to the manganese, and she ceased to attend on January 20, 1868.

In this case it appeared to me that the recovery of strength and return of colour were as rapid as could have been expected from iron. She was by no means well when I gave iron with the manganese; but it seemed likely that I should soon lose sight of her, and I thought it my duty not to deprive her altogether of the known good influence of iron, lest the improvement should be only temporary.

In a third case of severe anaemia and chlorosis with amenorrhoea, great improvement followed the administration of manganese, but twice the treatment was interrupted, and iron given, by my clinical assistant in my absence.

In another case, a very severe one, the manganese seemed to do no good whatever, and the patient was admitted into the wards, where she recovered under the usual treatment. It is sufficient to note the failure; the details would possess no interest.

Manganese was also given with good effect to children whose condition seemed to indicate iron.

Nickel.

Sarah C., aet. 20, servant. Ailing for six months. For four months suffering from cough. Catamenia not right for several years and absent for five months. Anaemia extreme, and attendant symptoms severe. Venous murmur loud. A systolic aortic murmur heard. No physical signs of disease of lungs.

January 2, 1868. Nickel chlorid. gr. ij., acid. hydrochlor. dil. ♏vj., infus. quassiae ℥j. t.d. Improvement decided and continuous. February 6. Colour better. February 27. Venous murmur scarcely audible; no aortic murmur. Last attendance on March 12, when she considered herself well.

Sarah P., aet. 29, servant. Anaemic; catamenia scanty; leucorrhoea. Thyroid body enlarged. January 20. Nickel chlorid. gr. ij. in infus. calumb. February 10. Feels quite well. February 24. No leucorrhoea. Catamenia more abundant. Ceased to attend.

I had many of those cases which make out-patient practice so unsatisfactory, the patients discontinuing their visits after one or two weeks. In some of these there seemed to be marked improvement, in others not. One patient gave me time to try manganese, nickel, and iron in succession, and derived no benefit from any of the three metals.

The administration of manganese with iron has long been practised, and, according to Pétrequin and other French writers, with great advantage; but I may mention one or two cases in which I have given nickel and iron.

Mary Ann B., aet. 22, servant. Ailing three or four years. Catamenia scanty. Markedly anaemic, and suffering from pain in the side, palpitation, and breathlessness on the slightest exertion. Venous murmur heard.

January 13, 1868. Mist. ferri c. quassia ℥j., nickel chloridi gr. i. t.d. Improvement very decided and uninterrupted. March 13. Well.

Caroline M., aet. 17, servant. Anaemia with usual symptoms. Catamenia absent; profuse leucorrhoea. Pupils remarkably large.

May 25, 1868. Mist ferri lax. (i.e. sulphate of magnesia with sulphate of iron), ℥ j. nickel chlorid. gr. ij. t.d. Leucorrhoea much better June 4. Improvement still greater June 15, when she ceased to attend.

Zinc.

This metal is mentioned, because I tried it under the impression that it belonged to the iron group. It failed altogether to effect any improvement, and I discovered, on referring to modern works on chemistry, that it is the centre of a group of which magnesium

and cadmium are the other members, and is not to be classed with iron.

The amount of evidence adduced is not great, but it should be remembered that the point to be examined was not whether manganese and nickel were superior or equal to iron in the treatment of anaemia, but whether these metals had any influence at all in this condition; and I am able to say distinctly that I have seen the strength improve and the colour return under their use, and this without the advantages of warmth, rest, and superior food afforded by admission into the wards of an hospital. Some of the cases I should not have thought of treating as out-patients had I not wished to employ them experimentally.

In using these metals as accessories to iron, it has seemed to me that *manganese* had a special influence in promoting the return of the catamenia, and *nickel* a special property of checking leucorrhoea. Manganese is much better borne by the stomach than nickel, and causes no disturbance when given in doses of 7 or 8 grains, beyond which I have not thought it necessary to go. Nickel usually causes vomiting, sooner or later, in doses above 2½ or 3 grains, and sometimes 1 grain added to the mist. ferri. c. quassia of the hospital produces this effect.

Postscript.—Chromium, another member of the iron group, is now under trial. In all cases improvement has followed. It has been administered in the form of chloride with a few drops of acid in water.

USES OF PHOSPHORUS

To further illustrate the hypothesis that "chemical groups form therapeutical groups" Dr. Broadbent read a paper on "Cases of Skin Disease treated by Phosphorus" before the Clinical Society in 1871, in the course of which he says: "Arsenic having a therapeutic action so well defined and so open to observation, it became an interesting question to ascertain how far similar powers were possessed by phosphorus and antimony. Antimony has been and still is employed in skin diseases, and it is given to horses by grooms to render the coat glossy. I have little personal evidence as to its effects to offer, as I preferred to employ my limited opportunities of observing skin diseases in trying phosphorus. This I have administered dissolved in oil." He then relates several cases of *eczema* and *psoriasis* successfully treated with phosphorus.

In the *Practitioner* in 1873 in a paper entitled "Clinical Illustrations of the Value of Phosphorus in certain forms of Disease of the Nervous System" he says: "When I gave phosphorus to out-patients at St. Mary's Hospital, the subjects of my earlier experiments, it was dissolved in almond or olive oil, and suspended in mucilage, in which it remained unchanged for a time. But this forms a nauseous mixture, and oxidation slowly takes place. Since that time I have given it in the form of pills with some extract, and sometimes dissolved in cod-liver oil. This last medium, however, is apparently the most treacherous of all, since in the brown oil the characteristic odour of phosphorus disappears at once, showing that oxidation has taken place. Pills also must undergo rapid deterioration. Fortunately a method has recently become available by which phosphorus can be given in a form at once active and inoffensive, namely, dissolved in oil or lard, and enclosed in a gelatine capsule; the dose is about $\frac{1}{30}$ of a grain, and it may be taken two or three times a day, always after food."

He then relates cases of *epileptiform vertigo*, *neuralgia*, and *neurasthenia* successfully treated, and in another paper in the *Practitioner* in 1875, two cases of *anginoid pain*, one of *pernicious anaemia*, and one of *leucocythemia*, all much benefited by phosphorus.

ON THE RELATION OF PATHOLOGY AND THERAPEUTICS TO CLINICAL MEDICINE

Portions of Presidential Address to the Clinical Society in 1887, *and of an Address to the Royal Medical Society of Edinburgh in* 1895

THE medical man is the intermediary who gathers up and applies for the benefit of the patient all the knowledge available at any given period, and whatever is most essential to individual members of the profession in the exercise of this function is the most worthy of attention. What is most important at the bedside ?—diagnosis ! But diagnosis is a word of deep meaning—thorough knowledge. It is no diagnosis at all to say that such and such is a case of valvular disease of the heart, or even that it is a case of aortic or mitral disease, obstructive or regurgitant. We must be prepared to estimate the degree of obstruction or the amount of regurgitation, to say whether the valvular change is probably stationary or likely to be progressive, to measure the effects on the cardiac muscle and cavities, and to judge in what direction and in what degree lethal tendencies are developing themselves. It is no diagnosis again to say that a man has hemiplegia ; we must localize and specify the lesion which has given rise to the paralysis. But the lesion is itself only an incident in some general morbid tragedy which is being acted in the body, and this must be read from the beginning.

Diagnosis, moreover, embraces a knowledge, not only of the seat and character of the disease, but also an appreciation of the condition of the patient in relation to the attack. Is the disease merely the winding up of a bankrupt constitution ?—a mode of dying rather than a cause of death ?—or is it, on the other hand, a necessary though violent readjustment of the organism to the environment with which it had been long getting out of harmony, or a defensive reaction against some powerful disturbing influence ? The problems presented are often of the highest degree of complexity, and we cannot too diligently cultivate our powers of observation, aiding the senses by the instruments with which science has furnished us, and bringing everything, as far as possible, to

the test of measurement and the balance; the exceptional cases of to-day are the common ones of to-morrow. There is no one who has for many years been in the habit of noting all he sees, who cannot look back to cases which he did not at all understand at the time, but which are now perfectly clear. And I would remind my hearers of the profound saying of Sir James Paget, "That an exception to one rule is only an example of another rule as yet unknown." The explanation of an exceptional case may thus be the revelation of a new law.

Value of Observation.—The basis of all clinical faculty is observation—that is to say, the intelligent and instructed employment of the senses to obtain facts—and in close relation with observation is inference, the right interpretation of the facts of observation. It is by observation that we obtain a more or less complete and accurate mental picture of each case of disease which comes before us, and the more clear and profound our knowledge of the influence of remedies, the greater will be the necessity of full and accurate observation.

What good, for example, would be a full theoretical and practical knowledge of diuretics, and of other methods of draining off fluid from the peritoneal cavity, to a man who was capable of mistaking fatty accumulation in the abdominal parietes and omentum for ascites? You will say I am supposing a fanciful and impossible case, but this is not so. Patients have been sent into hospital for operation, and I have been consulted more than once as to the necessity of paracentesis of the abdomen on account of ascites, which had resisted the most varied and energetic treatment—of course, with all the newest drugs—when the abdominal enlargement was entirely due to fat. On giving the flank a slap, the tremulous vibration of the flabby subcutaneous deposit could be followed across its surface to the opposite side, and this had been taken for a fluid wave. One had only to take up a great handful of the fat anywhere over the abdomen to demonstrate the condition.

To observe, means primarily to see, to use the eyes; and the faculty of seeing things is the essential endowment for the purpose of diagnosis. To this physical examination, the chemical and microscopical examination of the secretions and of morbid products are subsidiary. But in the zeal to acquire skill in percussion and auscultation, and in the use of the various "scopes," the exercise of the sense of vision is neglected. In all cases the information obtained by the sight is most important; in many it gives the clue to the diagnosis. It is often possible to recognize the disease at a glance. Not to speak of affections so conspicuous as Bell's facial paralysis, or the perfectly

distinct form of facial paralysis associated with hemiplegia, which when present without loss of power in the limbs constitutes facial monoplegia; or of appearances so characteristic as the projecting frog-like eyes of Graves' disease; or the heavy, dull, bloated, expressionless features of myxoedema; or of such obvious phenomena as the throbbing carotids which reveal aortic regurgitation—an experienced observer will distinguish at once between the pallor of pernicious, chlorotic, and ordinary anaemia; will recognize renal disease, or lead poisoning, or syphilis; will differentiate the appearance and expression of typhus and typhoid fever, of pneumonia, and other acute diseases; and, even in the absence of tremor, will recognize paralysis agitans from the fixed and sad expression of the face—not, of course, that he will rely in any single instance on conclusions so formed without careful and exhaustive inquiry.

Now, this faculty of seeing things, even in those originally gifted with observant eyes and minds, can only be arrived at by persevering cultivation, by consciously directing and compelling attention to minute points. It is not enough to take elaborate notes of cases in hospital; the countenance, the expression, the colour, the attitudes should all be studied; and time is well spent in looking intently at patients, especially such as illustrate well-marked examples of disease, till the portrait is fixed in the memory.

Inspection of the Chest.—Let me remind you again how important a place inspection occupies in the examination of the chest. The rounded barrel-shaped chest of emphysema, with the absence of expansile movement, the inspiratory retraction of the intercostal spaces and supra-clavicular fossae, and the prolongation of the act of expiration, give us at once not only a diagnosis, but the most important elements of our prognosis. A slight flattening or falling in above or below the clavicle on one side, or, without this, a slight deficiency of expansile movement, will often call our attention to minute auscultatory signs which might otherwise have escaped us, and so enable us to detect tubercular mischief at an early and perhaps curable stage. On the other hand, extreme immobility of one side of the chest, so that in respiration the other side almost seems to pivot upon it, may completely modify the interpretation to be put upon dullness, tubular breathing, and other signs of condensation and cavitation, and show that these changes are not necessarily the result of phthisical destruction of the lung, but in great part, sometimes altogether, of antecedent pleurisy.

Equally important is the information afforded by careful inspec-

tion of the cardiac region. To say nothing of the situation and character of the apex beat, when this is visible, or of the right ventricle impulse, which, when corroborated by palpation, throw so much light on the relative size and strength of the two sides of the heart, and on the degree of stress imposed upon one or other ventricle by conditions of the valves, or of the pulmonic or systemic peripheral circulation, the drag on intercostal spaces may be almost the sole evidence of adherent pericardium, and is very frequently the indication which clinches the diagnosis. It is astonishing how often adhesion of the pericardium, a complication which frequently determines a fatal issue of valvular disease, remains unrecognized for want of intent scrutiny of the praecordium.

Paralysis of the Diaphragm.—Two other respiratory phenomena I should like to mention, chiefly because they so often, according to my experience, escape attention. One is the reversed action of the diaphragm when this muscle is paralysed and flaccid. This may happen in the course of diphtheritic paralysis, or sometimes in peritonitis, or again from fatty degeneration of the diaphragm, which has importance as it may be associated with fatty degeneration of the heart; from disease in the cervical cord paralysing the phrenic nerves, or, rarely, from implication of both phrenics in some form of intrathoracic growth; or, again, there may be what is apparently a functional, and is certainly a temporary, paralysis of the diaphragm, aggravating terribly the dyspnoea of asthma, sometimes simulating asthma so far as respiratory distress is concerned, without, of course, the wheezing caused by occlusion of the minute bronchi.

Nothing is more simple than the recognition of this condition. Instead of the simultaneous expansion of the chest and advance of the abdominal walls in inspiration, it is seen that, as the thorax expands the abdomen falls in, and in expiration, as the chest returns from its expansion the abdomen suddenly bulges out. The two hands, placed one on the chest, the other on the abdomen, instead of rising and falling together, play at see-saw; and, as the thoracic movements are greatly exaggerated and the abdominal movements are very abrupt, in consequence of the diaphragm flopping to and fro like a wet rag in obedience to the changes of pressure, the see-saw alternation is very striking. Sometimes reversed diaphragmatic action is called thoracic respiration, but the two are quite different in causation and in significance; in thoracic respiration the diaphragm is fixed in one position or another, and not flaccid. An important diagnosis may turn on this distinction.

Cheyne-Stokes Respiration.—The other respiratory phenomenon I had in my mind is Cheyne-Stokes breathing. This is as common as it is interesting, and, so to speak, it imposes itself upon our notice, and yet it escapes observation with incredible frequency. One reason no doubt is, that the patient often employs the period during which the act of breathing is actually suspended in answering questions or in conversation, and appears to talk himself out of breath, so that the exaggerated respiration which gradually follows seems quite natural. Cheyne-Stokes respiration should be looked for in all cases of renal disease in which serious symptoms have supervened, and indeed in all patients, who are the subjects of old-standing high arterial tension, who complain of shortness of breath.

The Abdomen in Peritonitis.—The abdomen, perhaps, is subjected to more careful visual examination than the chest; and for this very reason, and also because I should have to go into wearisome details, out of place on such an occasion as this, I shall confine my remarks to one important instance of its use.

I might mention, as rare and interesting cases, one in which the upper end of the linea alba was dragged to one side at each inspiration from fixation of the right half of the diaphragm by a mediastinal growth, which gradually projected from under the lower end of the sternum into the right pleural cavity; and another, in which the right costal arch had sprung upwards and outwards, in consequence of the right pillar of the diaphragm having been dissected off from the bodies of the vertebrae by an aneurism; but the point in which inspection is of supreme importance in affections of the abdomen, is the extinction of the abdominal respiratory movements in peritonitis or enteritis. However great the distension of the abdomen by ascitic fluid, or ovarian tumour, or gastro-intestinal gases, in the absence of inflammation there will be some attempt at diaphragmatic action, recognizable by movements of the abdominal parietes; whereas, even localized peritoneal inflammation without distension, as in perityphlitis or pelvic peritonitis, will arrest all respiratory movement in the lower segment of the abdomen, and greatly diminish it above the umbilicus, often rendering it almost imperceptible. In general peritonitis, or in enteritis which has extended from the mucous to the muscular coat of the intestine (without of necessity any implication of the serous covering), the abdomen is absolutely motionless, as it also is in the case of abscess between the liver and the diaphragm. There is a reflex defensive fixation of the diaphragm in order to keep the abdominal viscera at rest. It may perhaps be well to add, in parenthesis, that opium or morphia, which are so constantly given in cases of peritonitis and of intestinal obstruc-

tion, may tend to confuse the issue and render the diagnosis between these conditions less clear. On the one hand, it lessens the respiratory abdominal movements, when there is distension without inflammation; and on the other, it may modify the inhibition of the diaphragmatic action, and allow of slight movement.

Let me relate a striking case which came under my observation not very long since, illustrating the value of the indications upon which I have just dwelt.

A gentleman, aged 45 or 46, had been exposed to severe cold. Three days later he was astonished by an unusually copious and soft action of the bowels, his habit being constipated. He had no further discomfort till after his dinner, which he enjoyed, when he began to suffer from pain in the abdomen, and was sick. In the course of the night the pain became so severe that morphia had to be given hypodermically. From this time there was absolute constipation and gradually increasing distension of the abdomen, with considerable pain, which, however, was mitigated by small doses of opium, but, for three days, no sickness. On the morning of the fourth day vomiting returned, and the matters raised were stercoraceous in character. The sudden onset, the absolute constipation and rapid distension of the abdomen, had given rise to a suspicion of intestinal obstruction, more especially as the general symptoms of peritonitis were not well marked, and the occurrence of stercoraceous vomiting seemed confirmatory of this idea. The question of an exploratory operation was consequently raised. There were, however, two indications which showed conclusively that the obstruction was not mechanical, but, so to speak, physiological, i.e. the result of inflammation. In the first place, the abdomen was absolutely motionless, and the respiration purely thoracic, which would not have been the case had the distension been due to obstruction; and, in the second, the abdominal parietes being extremely thin, some of the distended coils of small intestine defined themselves on the surface, and, when the abdomen was handled in the course of the necessary examination, these coils remained perfectly passive. Now, in intestinal obstruction, unless the bowel has been paralysed by large doses of opium, peristalsis is inevitably set up by manipulation, and can be seen in any coils which show on the surface of the abdomen. We had therefore a second trustworthy indication of inflammatory paralysis; the diagnosis of enteritis was thus perfectly clear, and a favourable prognosis was justified in the course of a few days.

Therapeutics.—It is not easy to deliver my mind on the subject of treatment. Nursing, care, appropriate food and stimulants do much for our acute cases; diet, clothing, climate, hygiene, are powerful

remedial agencies in chronic disease, and we cannot too closely study the surroundings of the patient, or direct too minutely the management of the sick room; we cannot inquire too carefully into the habits and mode of life which have led to chronic ailments or give instructions too detailed as to the means by which the injurious influence of past errors may be counteracted in the future. Were we to neglect these particulars, we should in effect ignore the first principle of rational treatment—"the removal of the cause"—and wilfully leave an insuperable obstacle in the path of recovery. But when we have attended to everything of this kind, we should be shorn of our strength, were we debarred from bringing to the aid of sufferers the powerful remedies we hold in our hands. In acute disease they often turn the scale in favour of the patient, as, for example, when death is averted in enteric fever by a timely dose of opium or quinine, or when the distended right heart is relieved by a calomel purge. In chronic affections, again, even if the patient is prepared to renounce his self-indulgence and forego his pleasures, or to neglect his business and abandon his ambitious schemes—and how often is he unwilling to do the one, or unable to do the other?—the long chain of evil consequences would be very slowly unwound, whereas we can often snap it almost at a stroke.

The mental depression, for example, which is often clearly traceable to functional derangement of the liver (to employ a term sanctioned by the authority of Murchison)—is it a reflex disturbance or inhibition of some nerve-centre, or is it the effect of a blood impurity acting as a poison to the brain like alcohol or opium? or is it a local or general derangement of the cerebro-spinal circulation? It is an everyday experience that a dose of blue pill will relieve the mind, clear the complexion, and relax the arterioles. Now, in many cases of obstinate and severe melancholia there is every reason to believe that the cause is outside the nervous system; and if we could, in the familiar examples of low spirits and irritable temper, trace accurately and minutely the way in which the initial gastro-hepatic disorder produces its effects on the nervous system, and the exact process by which these are reversed, we might understand and prevent or remedy the more serious overthrow of the mind seen in melancholia. It is not a final explanation, discharging us of all responsibility in searching out the cause, and rendering futile all efforts in the direction of cure, to find a family history of insanity. The hereditary tendency to melancholia may be indirect, and capable of defeat, just as hereditary tendency to apoplexy may be traceable to family gout, which can be prevented from developing its evil effects in individual members.

We look to quinine in ague, to mercury and iodide of potassium in syphilis, to iron in most forms of anaemia for effects as certain, and as constant as chemical reactions. These and similar examples, of themselves, or the single instance of the multifarious benefits obtainable from the administration of opium and its alkaloids, would justify us in asserting that we are able to modify profoundly and beneficially the course of disease. It is only want of knowledge of the morbid process on the one hand, and of the mode of action of the drug on the other, which prevents our having the same certainty in a thousand other instances. In proportion as our investigation of the cases which come before us is thorough, and our aim and object in the employment of medicines are clear, definite, and precise, in that proportion will our confidence in the uses of drugs be firm and will continue to increase.

With regard to the effects of remedies, however, we have, for the most part, only the testimony of experience, pure and simple, and our experience is a very insecure anchorage for belief, and a very feeble agency in carrying conviction to the minds of others. A man's own experience will, for himself, outweigh that of any number of others, and a single instance in which his own observation contradicts, or appears to contradict, the concurrent testimony of the rest of the world, will overthrow his belief in that testimony.

Mode of Action of Drugs.—The remedy for this is a knowledge of the mode of action of drugs. Already we employ digitalis with greater confidence and discrimination from the light which experiment has thrown upon its action on the arteries and heart, and nitro-glycerine, amyl nitrite and the nitrites have actually come into use on the ground of their experimentally demonstrated effects in relaxing the arterioles, and have proved to be of immense benefit. We have also introduced into practice, on experimental evidence, substances which directly and constantly bring down the temperature of the body. Enormous importance attaches to the investigations which are in progress on all hands, and I would refer particularly to Dr. Sydney Ringer's experiments, showing the influence of minute proportions of soda, potash, and lime salts on the action of the heart, which bring into evidence once more the radical differences which exist between substances commonly regarded as all but identical. Soda and potash, for example, are, for the most part, looked upon simply as two alkalies, which may be employed almost indifferently, the only exception recognized being, perhaps, that potash is the better solvent for uric acid and more of a diuretic, whereas their alkalinity is almost the only property they have in common ; when regarded as medicines their

relations with the organic constituents of the body and their influence on muscular contractility are totally different; they are found in different structures, and a minute proportion of any potash salt injected into a vein paralyses the heart, while soda salts have no such effects.

Antipyretics.—But while the mode of action of drugs is engaging attention, and great discoveries are bound to come before long, the progress made as yet is only small; and although the eagerness with which new remedies, which come accredited by science, are seized upon by the profession is a hopeful sign—there is danger of present injury from their employment. It is a tremendous piece of knowledge that a given drug will certainly lower the temperature, but this does not by any means warrant its indiscriminate administration in pyrexia. There is the further question, by what processes the temperature is reduced, and what concomitant effects are produced.

Quinine, salicine, salicylic acid and its compounds, antipyrin and like substances, aconite, and many other drugs, agree in diminishing the body-heat, but this is the only effect they have in common. There is positive peril in acting simply on the knowledge that the temperature can be lowered. Are we certain, to begin with, that a high temperature is always mischievous? May we not, in checking it, be interfering with defensive or readjusting processes? Are we to assume that Nature does not know what she is about when she sets the heat-regulating mechanism for a higher level whenever things go wrong in the economy? To take a practical example. In enteric fever, in which the long-protracted pyrexia becomes in itself a distinct danger by its effects on the nervous system, by disintegration of the muscular fibres of the heart and the glandular structures, and by the strain on nutrition, I am convinced of the utility of systematic cold bathing by my own observation as by the testimony of such men as Brand, Liebermeister, Cayley, and Tripier (of Lyons); but I think, from what I have seen, that the repression of body-heat by means of salicylates, antipyrin, and the like in this disease, is very dearly bought. Nor can I see what we have to gain by suppressing the short sharp fever, say, of pneumonia.

Even with regard to the employment of salicine and the salicylates in rheumatism—remedies which came to us straight from the laboratory, and whose mode of action is expounded in chemical formulae by Dr. Latham—I think it well to raise a warning note. Properly employed, salicine and the salicylates almost rob acute rheumatism of its terrors and dangers but, given in routine fashion, as we should give an effervescing

mixture, they have seemed to me capable of doing serious harm. At any rate, I have seen deaths in rheumatic fever, of a kind quite new and strange to me, after prolonged administration of salicylate of soda every four or six hours. The pain and fever had never yielded, the drug had been continued in hope of this result, till, in course of time, warning was given by delirium, the pulse became more frequent and extremely weak, what has looked like a modified hyperpyrexia has set in, with flushed face and hot perspiring skin, and the patient has rapidly sunk, the temperature, however, never rising above 105° F.

I come back, then, to my point that, in order that we may employ antipyretics, or remedies of any kind, with confidence, we must know exactly how the effects produced are brought about. With regard to this we are, however, only on the threshold of an inquiry which must go more deeply into the relations between physiological processes and the substances which modify them than, as yet, we have any conception of, for our information is superficial.

Chemistry and Therapeutics.—When, however, we come to ask questions of chemistry, for the most part we can get no answer. It is not the fault of medicine that we do not understand the mode of action of remedies ; it is that chemistry and other sciences are not sufficiently advanced for our purposes. We present a fact so constant and apparently so simple as the rapid removal of a syphilitic gumma by iodide of potassium, and we ask for an explanation. Perhaps we have here the one precious instance in which some sort of chemical explanation can be given. It was, certainly, the removal of a great stumbling-block when Professor Odling many years ago pointed out why the alkaline iodides might be active remedies, while chlorides and bromides, closely allied chemically, had no corresponding effects. The reason is that iodides are less stable than chlorides and bromides, and, when carried by the blood to the tissues, the iodine may be displaced from its combination with potassium or sodium, and may promote the oxidation and destruction of organic compounds, just as chlorine does in the process of bleaching. It has since been found that actively growing protoplasm determines this decomposition of potassium iodide, and, apparently, what takes place when this salt causes the absorption of a gumma is that the protoplasm concerned in the formation of the growth sets free iodine from the iodide, which, in its turn, liberates nascent oxygen, and this oxidizes and disintegrates the organic matter of the gumma.

The Action of Poisons.—We ask in vain a thousand other questions of chemistry, of electrical science. Let us put one. A given dose of morphia will dull sensory impressions, will suspend all the

cerebral functions and produce absolute unconsciousness, will finally arrest the respiratory reflex and cause death; the proportion of the drug giving rise to this result present in the blood circulating in the nerve centres must be so small as to be scarcely capable of fractional expression.

Now it is an axiom in science that for every action there is an equivalent expenditure of energy. It is not by its mere presence in the blood or in the nervous centres, or in the gland structures that a poison or remedy produces its effects; there is some dynamic agency at work. In the case of such bodies as the powerful organic alkaloids, this can scarcely have any other source than chemical change in this substance itself, while in the case of inorganic salts, it would seem that they condition reactions between other substances. We know that carbonic oxide proves fatal by combining with the haemoglobin to the exclusion of oxygen, so that the blood corpuscles are no longer available as carriers of oxygen to the central nervous system, and the anaesthetic gases and vapours act much in the same way, their safety consisting in the fact that the higher centres concerned in the mental operations and sensation succumb to the privation of oxygen before the lower centres, which are the seats of the vital reflexes, and that they are easily displaced by oxygen when this is again freely supplied. Here the proportion of the anaesthetic agent is comparatively large, and the mode of operation is physical rather than chemical, the vapour-density actually counting in the comparative effects of different anaesthetics.

Prussic Acid.—I might name other actions more or less capable of explanation, but it is such effects as those produced by aconite, atropine, morphine, and prussic acid, in which a fraction of a grain will give rise to profound disturbance or destroy life, that we need to understand. Twenty years ago, or more, when science was not prepared to answer nor I qualified to put such questions as those relating to the action of poisons and remedies, looking at the fact that all powerful alkaloids contained nitrogen, and that all, or nearly all, of which the constitution as well as the composition was known, belonged to the class of amides, imides, and nitrites, in which an ammonium radicle had been robbed of successive atoms of hydrogen, I conceived the idea that this departure from a stable type gave rise to a chemical tension capable of producing effects, when brought into presence of the chemical tension which seemed to me to be required for the explosions of nerve-force.

It occurred to me that prussic acid—the most deadly of poisons, and, at the same time, almost the simplest of organic substances—

offered the best possible chance of ascertaining the exact mode of action of, at any rate, one drug.

Hydrocyanic acid is composed simply of a single molecule each of carbon, hydrogen, and nitrogen—there is no room for complex changes. The hypothesis I entertained was that, when it reached the nerve-centres, the dislocating influence of nitrogen found its opportunity, under the influence of the chemical operations concerned in the evolution of nerve-force, and the nascent carbon and hydrogen flying apart, appropriated the oxygen needed by the nerve-cells, death resulting from the consequent arrest of activity of the centres in the medulla necessary to life. I cannot say that I proved my point, but I have met with no more satisfactory explanation since, and I met with facts which have never failed to interest men when I have related them. For instance, prussic acid is not a deadly poison to frogs. Many times I injected as much as ten minims of the pharmacopoeal solution (one minim of which was fatal to a rat) under the skin of a frog, and as long as he was allowed to hop about in the room he appeared to be no worse for it. If, however, even at the end of half an hour I put him under a bell-jar with other frogs, the prussic acid he exhaled sent the whole lot into a state of torpor. My interpretation was that the evolution of nerve-force in the frog was not attended with sufficient chemical energy to explode a HCN molecule. Another curious fact which turned up was that nitro-glycerine, which proved fatal to rats, apparently by paralysing the nerve endings like curare, was a convulsant of terrible energy when administered to frogs.

For a full comprehension of the action of remedies we must know and understand the chemical actions and reactions which are taking place in nutrition, secretion and disintegration; the chemical changes which attend and condition the evolution of nerve-force and muscular action; the modification of the chemical processes which result from the administration of a drug, and the change in the composition of the drug itself, through which the energy is evolved by means of which it produces its effects. The future of therapeutics is thus in the womb of chemistry. What miscroscopy has been to anatomy and pathology, chemistry will be to physiology and therapeutics.

REMOTE EFFECTS OF REMEDIES

An Address delivered at the opening of the Section of Medicine, at the Annual Meeting of the British Medical Association, held in Brighton, August, 1886. *British Medical Journal,* 1886, Vol. 2

I HAVE found nothing better with which to occupy your attention for a short time than a few illustrations of the desirability of looking beyond the immediate results of treatment, and of the necessity of exercising forethought in the employment of remedial measures, examples in fact of some remote effects of treatment.

That present relief is often obtained at the expense of future suffering and injury is a trite remark. I might take up my parable and speak of alcohol, the remote effects of which are so terribly frequent; but this is not a lesson which I need enforce, and I allude to it only for use as an illustration. If we were not so familiar with the exhilarating effects of wine, we should find it hard to believe that anything could so entirely lift a man above his surroundings, banishing sorrow and care and enhancing enjoyment, sharpening the wits and raising the spirits, conferring a feeling of self-satisfaction and throwing a cheerful glow on the most unfavourable circumstances. Supposing all this could be attained without speedy reaction and ultimate ruin to body and mind, is there any consideration which would prevent men from unlimited indulgence?

There will occur to all, again, the pain and depression, the bodily derangement, and the mental wreck, by which the opium-eater, or the devotee to hypodermic morphine, ultimately pays for his delicious dream or her factitious happiness; the miserable irresolution and wretchedness which, after a time, overtake the man who habitually resorts to chloral for the sleep which he ought to court by fresh air and exercise, and by the removal of the influences which give rise to sleeplessness. Even the bromides—the least injurious perhaps because least powerful of the remedies employed to procure sleep—besides producing the well-known acne, may bring a man into a state of childish dementia, which, fortunately, does not last. We have not yet begun to see the evil effects of the newer hypnotics, such as paraldehyde, urethane, etc., but coca or cucaine is already beginning to claim its victims, who

gradually develop an excitement which becomes maniacal, or an exaltation like that of general paralysis.

These glaring examples of injurious remote effects of measures which are immediately beneficial are only adduced in order to show the direction of my thoughts.

It may be said that the injury is the result of unauthorized abuse of remedies by patients, and of weak or vicious self-indulgence; but so also will many of the injurious effects I propose to consider be seen to result from patients taking the treatment of their ailments, real or supposed, into their own hands. The point is, that very frequently the treatment has been initiated, or at least sanctioned, by the medical man, just as he has prescribed or administered the first hypodermic dose of morphine.

Remote Effects of Gout Specifics.—Let me begin with the familiar subject of gout and some of the remedies which are employed in its treatment. It is well known that we can relieve the pain and cut short the paroxysm of an acute attack by the free use of colchicum, veratria, and other drugs having a similar action. It is equally well known that their injudicious use may produce great immediate prostration, and that the remote effects of their habitual employment are often most disastrous. We may, however, administer colchicum; we are, indeed, compelled by the frightful suffering to give it freely in some cases; but we are justified in resorting to it only on condition that we do for the patient by after-treatment —aperients, alkalies, iodides, etc., with careful regulation of the diet—what the attack would have done; that is, clear the system of gout poison, and bring the secretory and excretory organs into a state of functional efficiency. Or the end may be attained by a visit to one of the many baths and waters known to be efficacious in gout.

These precautions, and the self-denial required for continual immunity, are frequently unacceptable to the gouty convalescent, and are disregarded; and, when gouty pains return, they are kept down by the remedies which have been found to give relief during the attack, or by some so-called specific. This may be done with success for a very long time. Meanwhile, however, the remote effects are in preparation. These are mainly due to imperfect elimination. It is not implied that colchicum and its allies directly arrest the elimination of uric acid existing in the blood; it is rather antecedent metabolism which is checked, and nitrogenized waste other than urea and uric acid accumulates in the blood and tissues. Such impurities provoke resistance in the arterio-capillary circulation, and we enter upon the condition of high arterial tension, with all its varied damage and danger.

It can scarcely be too often repeated that all the effects upon the arteries on the one hand and the heart on the other, associated with contracted granular kidney, namely, arterial degeneration, cerebral haemorrhage, aneurism, dilatation of the left ventricle, and valvular disease, are just as commonly produced, independently of kidney disease, by high pressure in the arterial system due to other causes; and such pressure in an extreme degree may be induced prematurely by the means taken to keep down gout. Even convulsions not distinguishable from those occurring in uraemia may be induced. A case which occurred in my experience within the last twelve months may illustrate effects of this kind.

A man, aged 37, living a sedentary life, of strictly temperate and indeed abstemious habits, so far as could be ascertained, consulted me in October, 1885, on account of continuous headache, with weekly exacerbations of frightful severity, at times attended with vomiting. I was at once struck by the high tension in the arteries; the temporals were prominent and tortuous, the radial was large, full between the beats, and compressed with difficulty, and the pulse was long. The urine, an examination of which was at once suggested by this state of the circulation, was found to have a good colour and normal specific gravity, and did not at this time contain albumen, although traces were found subsequently. I looked upon the paroxysmal headaches as of migraine character, and did not attach sufficient importance to, or adequately deal with, the high arterial tension, especially as the arteries are tightened up in migraine; and I gave only a mild blue and colocynth pill twice a week, while arsenic was prescribed with carbonate of potash, for the migraine. Three weeks later, the paroxysmal headache was better, but the continuous headache remained; no important change was made in the treatment.

Nothing more was seen of the patient until I was called to meet in consultation Dr. Wilbe, who had only been summoned to him on the supervention of urgent symptoms. These were, pain in the head of overwhelming severity, incoherence, stupor, intolerance of light and sound, and violent muscular jerkings. He lay on his back or side, the chin down on the breast, his arms bent, the knees drawn up, and all the muscles of the chest and abdomen in a state of powerful tonic contraction, while every three or four seconds there was a violent momentary spasm of the abdominal and thoracic muscles, attended with synchronous flexion on the body of the arms and legs. The heart was beating violently, and the tension in the arteries was extremely high, the radial feeling like a solid incompressible cord. (I

may say, in parenthesis, that I have never found the pulse so strong and incompressible in uraemic convulsions as in this patient, the heart usually appearing to have failed in vigour when they supervene.) It seemed as if the vessels must burst under the strain, and the symptoms were obviously in relation with the high arterial tension; venesection was therefore practised, and sixteen ounces of blood were withdrawn; this was followed by a calomel purge. Great, but not complete, relief was at once experienced, and the patient slowly recovered. A larger bleeding would have been more effectual.

The extraordinary tension present in the arteries throughout this case was incomprehensible until, one day, the patient's sister produced a number of empty bottles, which had contained Laville's specific for gout. He had, in fact, had pain which he attributed to gout and, finding that he was relieved by the specific, he had taken it largely.

Some of the most striking illustrations of injurious remote effects of gout-specifics are furnished by London painters and plumbers. Lead in the system, we know, is a common cause of gout, apparently through its interference with oxidation and metabolism; and plumbers and painters are, in consequence of absorption of lead, extremely subject to this disease. There are, however, well-known gout pills which are very effectual in relieving the pain, and they are taken, as a matter of course, whenever gouty symptoms set in. The attacks being prevented or suppressed, imperfectly oxidized matter, which might have been burnt off, accumulates in the system; and it is among workers in lead who have resorted to this practice that we see enormous deposits of urate of soda in the hands and feet, and find the arteries ruined by degenerative changes, the result of protracted high pressure, or the heart dilated by the resistance in the peripheral circulation, contracted granular disease of the kidney, itself a product either of the state of the blood or of the vascular tension to which this gives rise, often aggravating the effects of the original blood condition.

I need not enumerate the disasters which attend the structural changes mentioned—cerebral haemorrhage on the one hand, dropsy on the other, acute pulmonary and other attacks, uraemic convulsions. Let me add, however, that it is not only in these direct and conspicuous consequences that injury is manifested; diseases, which would otherwise be attended with no danger, prove fatal when the heart is worn out by the constant struggle with the resistance in the arterioles and capillaries, and convalescence is slow, difficult, and imperfect when the vessels are deteriorated. In my experience, too, I may mention that

gangrene of the lungs has been disproportionately frequent among workers in lead, who have suppressed gouty tendencies by the means named, and it is easy to understand a liability to this complication. Pneumonia is one of the local inflammations to which the constitutional condition may give rise; the circulation has not the normal freedom and rapidity, and there is, moreover, a tendency to coagulation in the blood of gouty subjects, as is manifested by the familiar thrombosis of veins. Such coagulation in the vessels of a pneumonic patch will be the determining cause of gangrene of the lung.

Effects of the "Banting" Diet.—There are other ways in which abnormally and injuriously high arterial tension may be encouraged. One of these is the "Banting" treatment of obesity, as understood and practised by the public. This consists essentially of a highly nitrogenized diet, and, indeed, in the exclusion of the non-nitrogenized sugars and starches, together with a limitation of the amount of fluid taken as drink. We have, therefore, at the same time, a disproportionate amount of nitrogenized matters introduced into the system, which have to be broken up, oxidized, and excreted, and a diminished supply of water, which is essential to active metabolism and elimination. An important part of the method, when properly carried out, is exercise, which, in fresh air, may work off the impurities; but this element of the treatment is usually neglected. The amount of fat is undoubtedly often reduced, but the imperfectly oxidized nitrogenized waste present in the blood provokes resistance in the capillaries and generates tension in the arteries.

Cases have repeatedly come under my notice in which the injurious effects, mostly cardiac, of this condition have been manifest; and, in one instance, the first symptoms of dilatation of the left ventricle, which ultimately proved fatal, were so distinctly traceable to the period at which the Banting diet was adopted, that no doubt was left in my mind that it had had a preponderating share in producing this change in the heart.

Gouty Diabetes.—The examples cited, it may be said, simply illustrate the folly of violent interference with natural processes by people who are ignorant of the consequences; but sometimes the colchicum treatment of gout, or the Banting process, is initiated more or less under the advice of medical men; and, in my opinion, a similar error, for which medical teaching is directly responsible, is committed when strict dietetic treatment is insisted upon in the glycosuria of stout, elderly, gouty subjects. I will not stop to discuss the question whether this is to be called diabetes or not; but, to be quite clear, I may state that the cases I have in mind are

not those in which there is merely, from time to time, a small amount of sugar in the urine, but such as have copious urine with high specific gravity, and the constant presence of much sugar, often with thirst.

Now, in some of these patients, the establishment of habitual and free elimination of sugar is coincident with the disappearance of many distressing symptoms, especially digestive troubles, loss of appetite, irritability of temper, disturbed sleep, and morning depression. The altered chemistry of the liver is, in some way, a relief; perhaps it is by the increased elimination of the urea which accompanies the sugar, the two coming from the splitting up of nitrogenized matters. I do not wish to argue from this that we must let the glycosuria alone, but only that we must bear it in mind in our treatment.

Now, in my experience, this form of glycosuria is almost always associated with high tension in the pulse, which is not constantly the case in the diabetes of early life, the pressure in the arteries here being often extremely low; as every one knows also, it is common in gouty persons, and it may or may not appear to relieve the gout; very often it does nothing of the kind. It appears to me that, with all these evidences of the presence of nitrogenized waste in the blood, it can scarcely be right to insist on a diet from which non-nitrogenized matters are strictly excluded. It may be added that, as a matter of practical observation, such a diet is rarely, if ever, adhered to; patients belonging to this class are not usually such as have been accustomed to self-restraint, or such as can resist the solicitations of a newly reviving appetite; and, when it is attempted, do we not find the mouth to become foul and dry, the appetite fail, the bowels deranged, till we are compelled to relax the restrictions?

But this is not all; here again, I have met with instances in which the heart has suffered, the long full pulse of sustained tension being replaced by the sudden and short wave in a large full artery, indicative of dilatation, corresponding signs being found on examination of the heart.

These are the cases in which cures are effected at Carlsbad, Marienbad, Vichy, and elsewhere, by the eliminating action of large quantities of water, aided, more or less, by the saline matters contained in it. We have thus therapeutic evidence that what is needed is elimination.

Now, our object is not the removal of the last trace of sugar from the urine, but the comfort, happiness and long life of the patient. With regard to the prolongation of life, it is impossible to speak definitely; but I can say without hesitation that the patients

feel better and happier under eliminant treatment, with a modified mixed diet, than under a rigorous exclusion of starchy foods. They seem, moreover, to go on living indefinitely.

The treatment I have in my mind is the administration of a blue and colocynth pill once or twice a week, followed up by salicylates and alkalies. It may be added that, although this is not the effect chiefly aimed at, very commonly sugar is completely banished from the urine for months, and sometimes for years.

Effects of Asthma Cures.—Another example of injury, gradually wrought by a remedy which affords alleviation of symptoms, is furnished by asthma. There are certain powders, of which "Himrod" seems to be the most popular, the fumes of which, when burnt, sometimes give speedy relief to the asthmatic paroxysm. We cannot wonder that sufferers should resort to this or to any other expedient which promises to cut short the frightful suffocation of the attack; and, in some cases of the purely neurotic asthma, no great harm is done. The bronchial spasm, or hyperaemia, is a trick of an excitable nervous system, and may be met by a remedy which satisfies the demand for a local stimulant or sedative; but the solanaceous drugs, of which the powders are largely composed, contain powerful alkaloids, which obtain direct access to the blood through the pulmonary capillaries, and produce their effects on the nervous and vascular systems; and, when we see the stupefied mental condition, and the congested face and eyes, and note the large, weak, sluggish pulse, showing paralysis of the arterial walls, it is clear that such effects cannot be indefinitely repeated with impunity. It is where there has been a catarrhal element in the asthma that the results have seemed to me most disastrous. The right ventricle loses its tone, like the muscular coats of the arteries, and, instead of becoming hypertrophied, yields to the resistance in the pulmonary circulation, and is dilated, finally giving rise to systemic venous stasis and dropsy. In one such case, in a young and otherwise strong man, seen with Dr. Andrews, of Hampstead, I had the opportunity of verifying this dilatation of the right ventricle post-mortem, there being no other discoverable cause of death. The asthma began in catarrh; the "Himrod," giving relief at first, was employed immoderately, the whole house reeking with it; acute dilatation of the right ventricle took place, and ultimately proved fatal.

Migraine.—Another affection which I have sometimes seen treated without regard to the remote effects of the treatment is sick-headache, so called. This is essentially a nervous paroxysm—a nerve-storm some are pleased to call it—the liability to which is usually inherited, the attacks having a tendency to recur at more

or less definite intervals, and usually becoming more frequent and severe when the general tone is low, but also being provoked by emotion, fatigue, certain winds or states of atmosphere, the catamenial period, derangements of the stomach or liver, etc. We cannot, so far as I know, abolish this liability. We can at the most reduce the frequency and severity of the attacks by keeping the general health at its best, and by maintaining a high tone of the nervous system, when exciting causes of an ordinary or average kind are successfully resisted. It is a significant fact, however, that with advancing years, and in women after the cessation of the catamenia, the sick headaches frequently disappear; and I have observed, too, that, with exceptional intellectual vigour in old age, the liability to paroxysmal headaches has been retained.

There is scarcely a week in which we are not called upon to treat these sick headaches, and we can do much to relieve the exacerbations which have become intolerable, and have driven the patient to seek our aid; but we must acknowledge to ourselves, and I think it is only prudent to acknowledge also to our patients, the limitations of our power. We may not, perhaps, so far reconcile the sufferer to his ailment that he will be rather proud of his migraine than otherwise, as is not unfrequently the case in France, where *ma migraine* is spoken of almost in a tone of affection, or is looked upon as a sort of distinction; but he will listen to common sense, and not be too ready to anticipate the senile immunity which we may promise him.

But the radical treatment of sick headache is sometimes seriously undertaken, occasionally from the point of view of cerebral congestion, more frequently when considered as a symptom of gastro-hepatic derangement. Not very long since, a lady came to my consulting-room for advice with regard to congestion of the brain, of which she said she had had several severe attacks. She had been confined to bed in a darkened room, and had had leeches and blisters applied, and, after recovery from the acute phase, had been placed on a most rigorous diet, from which she had never ventured to deviate. She was wretchedly thin and weak, and quite unequal to the care of her house, any emotion bringing on headache, which was looked upon as a threatening of renewed cerebral congestion. Inquiry satisfied me that the first attack of congestion of the brain was simply an exaggerated sick headache, and subsequent attacks were more severe in consequence of the treatment. I prescribed careful feeding up and arsenic, which was carried out by my friend Mr. Roche Lynch; and nothing more has been heard of congestion of the brain, while the patient has been restored to health and strength.

Without discussing the relation between migraine and the liver, or the feasibility of curing sick headache by removing hepatic derangement, let me relate a case in which this was attempted with apparent success, but with remote effects which more than counterbalanced the result.

A hard-working professional man, between 50 and 60, who had suffered from sick headache all his life, and from constipation, was told that the one depended on the other, and that a morning dose of Friedrichshall water and a daily enema would relieve him of both. In the course of months this came to pass, but he became subject to tinnitus aurium, and to attacks of giddiness, in which he sometimes fell down, while he was no longer equal to his duties. The vertigo was attributed to overfeeding and stomach derangement, and he was strictly dieted, but without good effect; while an ulcer of the cornea developed itself, which may or may not have been the result of innutrition.

I first saw him some time after the establishment of the corneal ulcer and heard only of the giddiness—nothing of the migraine and its treatment. The giddiness was indeed trying enough to monopolize both his attention and mine—he walked unsteadily, staggered on the slightest occasion, and had been known to fall down with momentary loss of consciousness; he was anaemic but not thin, and I found very high tension in the pulse with a weak left ventricle. Looking upon the vertigo as the result of imperfect supply of blood to the brain, and this as due to the inability of the heart to cope with the resistance in the arterio-capillary network, I endeavoured to meet these indications by the eliminant action of a mild blue pill with colocynth, which I relied upon to diminish the peripheral resistance, while I gave arsenic with nux vomica and quinine, to improve the condition of the blood and the tone of the cardiac muscle.

The immediate effects were most satisfactory. The feelings of insecurity and habitual giddiness were lost, and, although not free from all his symptoms, the patient felt equal to work. This, however, did not last long, for about six weeks later, after worry and strain, he again fell down unconscious. It was only after another severe attack of this kind that I discovered that, determined to retain the benefit he supposed himself to have derived from the Friedrichshall water and low diet—the loss of his headaches—he had persisted in these measures, defeating in this way the endeavour to improve his nutrition and increase his strength. From this time, under better food, there was no recurrence of the attacks, and a winter at Cannes completed the restoration to health and strength. I should, however, have more

confidence in the permanence of the recovery, as I told the patient, if he had a return of original headache.

Effects of Treatment of Indigestion.—This case leads up to the last illustration I shall bring before you of present relief purchased at the expense of subsequent injury to health. This is the treatment, by too careful and restricted a diet, of certain forms of so-called indigestion. There are numbers of people, both men and women, but chiefly women, who begin by having pain in the epigastric region, at a variable interval after eating, pain, associated or not with flatulence. This is attributed to some particular article of diet, which is dropped, with perhaps temporary suspension of the discomfort. The pain, however, soon returns and after a number of experiments medical advice is sought. It is not uncommon for the medical man to take the same view of the case as the patient, and a scale of diet is laid down with great minuteness, adopting perhaps all the sufferer's restrictions and adding more, which is observed with all the greater scrupulousness the more stringent its injunctions and obeyed all the more readily because the appetite is wanting. I have known this to be done even when neuralgia was crying aloud for more and better blood to be sent to the nerve centres.

There are still discomfort and a sense of repletion after food; and the patient, considering himself to be in possession of the secret of his cure, improves upon the instructions given him, and the list of permissible foods is still further cut down, till starvation point is almost reached, or acute illness puts an end to the dieting. I have seen such documents, with the pen run through one article after another of those permitted by the medical man; and a case came before me in the spring, in which the only food taken was soaked biscuit, and this not by a hysterical girl, but by a mother who was devoting all her energies to the care of her family. The aggravation of the pain, which had from innutrition become severe gastro-enteralgia, was attributed to the obstinacy of the indigestion, and I was expected to suggest something still more simple in the way of food. It was with difficulty that the patient was convinced of the connexion between her increasing suffering and her diminished nourishment; but, this done and the requisite measures adopted, she gradually recovered.

I cannot say that fatal results of such treatment have come under my notice, but I have met with many instances in which it has led to great weakness and suffering in the individual, and serious interference with the comfort and welfare of families. The energetic wife and mother has become a fretful invalid, migraine has been aggravated, other neuroses developed, in one

case the most severe anginoid paroxysms, and all capability of useful work has been lost. Sometimes an acute attack of illness supervenes, during which liberal nourishment is insisted upon, and the patient gets up comparatively well. The illness in such cases gets the credit of improvement in the general health, which is really due to the food given during its continuance and during convalescence.

A variety of this dyspepsia of which I am speaking is the winter indigestion of women and weakly men. As cold and damp weather sets in, there are many persons who begin to suffer from pain after eating and flatulence ; or these symptoms may not set in till later in the winter, when the cold and short days have reduced the vital powers. Very frequently, the connexion between the indigestion and the season of the year is not recognized, and the subjects of it simply look upon themselves as liable to dyspepsia, which they associate with certain articles of diet, instead of with the winter, or attribute to want of exercise and fresh air. As is well known, however, cold, and especially cold with damp, will inhibit digestion, sometimes so completely, that a hearty meal, eaten with avidity after a cold drive, will be vomited almost unchanged hours afterwards, but this takes place more frequently in a minor degree, sufficiently to give rise to discomfort and a sense of distension, or the cold will inhibit the hepatic functions, and cause constipation.

Now in all such cases, it is not the food which disagrees with the stomach, but the stomach which disagrees with the food ; and the appropriate treatment is not levelling down the nourishment to the digestive capacity of the stomach, but the bringing up of the functional energy of the stomach to the requirements of digestion, by extra food of a stimulating character, such as beef tea, or an egg-flip between meals, by stimulants at meals, and by tonics. So with regard to winter-indigestion ; winter is not the time for cutting off food, when it is required in larger amount to neutralize the influence of external cold. What is wanted is protection from the depressing influence of cold, or the means of neutralizing it.

It is quite true that most people eat far too much, and, again, that with regard to the stomach, as well as to all other organs and parts of the body, the principle of functional rest is of primary importance in dealing with disease ; and restriction of food, and even temporary starvation is often necessary ; but we must distinguish, and must not starve those who are suffering from inadequate nourishment, or employ treatment for catarrh, or ulcer, or organic disease, when nothing of the kind is present.

I had on my list a reference to the injudicious use of purgatives, and to the employment of aperients, without reference to the special

cause of constipation, and without discriminating between the differing modes of action of the drug selected. I might also have extended the catalogue of injurious remote effects of remedies, but it is time to make way for the more important papers which await your attention.

ANNUAL ADDRESS TO THE NORTH-WEST LONDON CLINICAL SOCIETY

Extract

Medical Rejection of Candidates for the Army

The Clinical Journal, 1897

I do not think I can do better than take up a subject which has recently been before the public in the *Times* and other newspapers —the rejection of candidates for the army and public services on the ground of unfitness in point of health.

In my judgment this has sometimes taken place on totally inadequate grounds, and I think it may be useful to illustrate from my experience conditions which have led to this—more especially since on similar inadequate grounds boys and young men are not unfrequently debarred from public school and university life, forbidden to engage in games of all kinds, and sometimes condemned to the wretched existence of a confirmed invalid. Occasionally, but much more rarely, lives are refused for insurance.

Irritable Heart.—One of the reasons assigned for rejection is "irritable heart." A young fellow of 18 or 19, who has passed successfully for Woolwich or Sandhurst, presents himself before the medical examiner in a state of extreme nervous excitement; his pulse is beating any number of times a minute, and, perhaps not quite regularly, and on examination the cardiac impulse is violent and extends over an unduly large area, perhaps lifting the sternum and giving rise to apparent pulsation outside its right border. Any proper appreciation of the sounds is impossible, and there may be murmurs at one or more of the orifices. The breathing will at the same time be short and hurried. But such disturbance of the action of the heart, however exaggerated, is not inconsistent with perfect soundness and efficiency. This is so far recognized that, unless there are valvular murmurs, the boy is usually told to come up again for examination in three or six months. But if he has to face the same medical examiner under similar conditions, conscious of the previous failure, and knowing that his whole future is at stake, the chances are that

his heart will again go off at a gallop. It may be said that a boy who is so nervous would perhaps be unequal to an emergency such as might befall him in a soldier's life; but, on the other hand, a highly strung, sensitive nervous system may be exactly the one which will rise to an occasion and render service of which a more stolid organization would be incapable.

In such a case the personal and family antecedents would be of extreme importance. If the boy has been good at football and cricket, distinguished in athletics, fond of hunting and shooting, has followed the beagles, there cannot be anything radically unsound about his heart. It would take a great deal to make me reject the captain of the football team of a large school. What frequently happens is that a boy intending to enter the army is taken from school, it may be because of his devotion to games rather than to work, and sent to a crammer's, where long and late hours of study, and restricted opportunities of exercise and fresh air, with possibly unlimited tobacco, impair his bodily vigour and increase his nervous susceptibility. A fair test would be to let a candidate, whose fitness for military duty is in doubt on account of irritable heart, run a mile round one of the cinder paths at his own pace, noting his time, but especially the period at which he got his second wind and how he finished up. Sometimes the simple expedient of letting the young fellow run up two flights of stairs will bring the heart to its senses—a physiological has been substituted for an emotional cause of acceleration of its action.

Murmurs.—But a more common cause of rejection than irritable heart is the presence of a murmur perfectly innocent of significance. One of such murmurs is the spurious murmur not unfrequently heard in the apex region, and to the left, which is produced by compression of the overlapping lung by the heart during systole. It simulates closely a soft systolic mitral murmur, but is really due to displacement of air in the lung. It is easily recognized, and distinguished from a valvular murmur by the fact that it is heard only during inspiration or while the chest is full; it is, in fact, an intensification of the inspiratory murmur. Why this pulsatile respiratory murmur should be heard in some people and not in others cannot be stated. It has no unfavourable significance whatever, and almost always disappears as the chest is developed, though it may be heard at any period of life.

Sometimes this systolic exaggeration of the breath-sounds is audible all round the left chest to the back, and indeed over a great part of the lung, but in this case there are usually pleural adhesions.

The *pseudo*-murmur just described has not in my recent experi-

ence been assigned as a reason for rejecting an army candidate; but I have known it to be looked upon as indicative of valvular disease requiring treatment by digitalis, and demanding all sorts of precautions in the matter of exercise. A murmur more frequently considered to be due to valvular lesion, but which has no such significance, is that produced in the pulmonary artery. Its maximum intensity is in the pulmonic area, the third left space half an inch from the edge of the sternum; but it may be audible at the apex, in which case it can be followed from the pulmonic area downwards along the left border of the heart to this point. When at all loud it is usually heard over a great part of the right ventricle, and therefore in the tricuspid area, and it may be audible so far across the sternum as to give rise to a suspicion of aortic obstruction. It is a most capricious murmur, often varying in intensity and character in the same patient; sometimes audible only in the erect position, more frequently making its appearance or intensified when the patient lies down. When it is heard only in the pulmonic area there is little danger of its being looked upon as serious, but its invasion of the mitral or aortic area is regarded with suspicion, and may easily be taken for an independent murmur. It is not, however, heard to the left of the apex, and it can usually be traced continuously upwards to the pulmonic area.

Another means of distinguishing it can also be applied, which arises out of its mode of production, and this makes it worth while referring to its causation. This has been much discussed, and various explanations have been given. I have no manner of doubt that its mode of production is as follows:—The conus arteriosus of the pulmonary artery is usually covered by a layer of lung which intervenes between it and the chest wall, but in a certain number of subjects the lung does not come sufficiently far over the heart, or the overlapping margin of the lung is very thin. When such is the case the systolic bulging of the conus brings its anterior wall into contact with the wall of the chest, and a slight flattening of its convexity takes place, and this gives rise to vibrations in the blood current within, which constitute the murmur. A proof of the explanation here given is obtained by telling the patient to take a deep breath, and hold it. A cushion of lung is brought over the conus, and the murmur disappears, and with it, of course, the extensions which have excited apprehension of valvular disease.

I have sometimes succeeded in eliciting a murmur of this kind, when it was not naturally present, by making the patient lie flat on his back, and exercising pressure over the pulmonic area.

The pulmonic systolic murmur is more common in women than in men, but it is frequently met with in boys and youths. It almost

always ceases to be heard when the chest is fully developed, especially when it is expanded by drilling and gymnastics; but it may in some cases be reproduced at any period of life by sustained and violent exertion.

I am tempted to transcribe from my notes a description of the murmurs heard in the cardiac region of a young man of twenty, captain of a public school football team, and distinguished in all forms of athletics. The apex-beat was a well-defined push a little outside the normal situation, and the right ventricle impulse was forcible. At, but especially to the left of the apex, was a systolic murmur, very distinct during inspiration, gradually subsiding during expiration, but not always absolutely disappearing. It could be followed round the chest, and was particularly distinct between the scapula and the spine at the usual spot, where it was audible at all periods of the respiratory cycle, though less so at the end of expiration. This seemed to be conclusive of the existence of mitral regurgitation. On further examination, however, the murmur was heard all over the left lung except at the apex anteriorly; it was distinct, though weak, in the supra-scapular region. But besides this there was another and quite separate loud rough murmur over the pulmonary artery, having its maximum intensity in the left third space about an inch from the sternum, which disappeared when a deep breath was taken; it was conducted upwards and outwards to the subclavicular region. Taken apart from the murmurs the heart-sounds were normal, and in particular there was no accentuation of the pulmonic second sound.

From previous experience I considered myself entitled to conclude that the left pleura was adherent, and it could be seen that the left half of the chest was narrower than the right, and that its movements in respiration were markedly less. Probably with general parietal pleural adhesion, there was adhesion of the lung to the pericardium.

I allowed this young man to play cricket and all other summer games, and to cycle. His wind and endurance were very good, and on examination after five months I found him none the worse for very vigorous exercise.

It will, of course, be understood that I am not now referring to the haemic murmurs producible at every orifice by anaemia. A state of bloodlessness competent to the production of aortic and pulmonic murmurs would be a sufficient ground for, at any rate, a temporary rejection of a cadet.

Other murmurs are heard in the *tricuspid area* which are perfectly harmless. One is not properly speaking a murmur, but a sort of systolic scratch, which on careful examination is readily recognized as exocardial. It probably corresponds with a white

thickened patch often seen in the pericardium over the right ventricle, attributable to local pressure and friction, especially when the lower end of the sternum is depressed. But a real tricuspid systolic murmur may be met with at any period of life, which prolonged observation, extending in several cases over many years, has convinced me to be quite innocent.

Tricuspid regurgitation, since it usually occurs as the last stage in a series of valvular lesions making for back pressure and stasis, is properly looked upon as an extremely grave affection, and thus disposes us to regard any reflux through this orifice with apprehension. A systolic tricuspid murmur implies such reflux, and if this were considerable in amount it would constitute an obstacle to the venous return and a disqualification for military service. In no case of primary tricuspid incompetence, however, have I found evidence of any appreciable regurgitation, and it will be remembered that tricuspid regurgitation is very easily detected by the jugular pulsation, enlargement of the liver, and distension of the right auricle to which it gives rise. This tricuspid murmur, which may be musical or blowing, and which may be heard along the lower border of the heart as far out as the apex, so as to be taken for a mitral murmur, is rarely constant; it may be present only in the recumbent or, much more commonly, only in the erect position. It may even vary during respiration, and become audible only towards the end of expiration. Not uncommonly it is obviously produced by distension of the stomach. I have come to attribute it to pressure on the yielding wall of the right ventricle, where it rests on the diaphragm, which happens to take effect at a point corresponding to the origin of a papillary muscle. The adjustment of the valvular curtains is thus deranged, and a minute leakage is permitted which gives rise to the murmur.

There are *mitral systolic murmurs*, the result of actual valvular lesion, which are attended with so little regurgitation that they might be disregarded, if it were not for the liability to recurrence of the rheumatic attacks to which they were originally due. The criterion is absence of displacement of the apex-beat and of accentuation of the pulmonic second sound, or undue right ventricle impulse, together, of course, with absence of symptoms.

Many times I have allowed boys to go to a public school with no restrictions as to games except with regard to football, house runs, and training for races, who had been condemned to a life of inactivity. One such boy, who had not even been allowed to walk upstairs, carried off the prize for gymnastics in the contest between the different public schools.

Dilatation of the Heart.—I must not leave the subject of the heart

without referring to dilatation. This is not uncommon in boys, especially boys who have engaged in very violent forms of exertion, or who have been at cramming institutions and have taken all their exercise for the week at one time. The apex-beat is outside and slightly below the normal situation, and is diffused over a considerable area; and, when the action of the heart is frequent, the first sound will be short. It is usually taken for granted that dilatation of the heart, when it is found to exist at all, is a fixed condition, and these hearts are looked upon as permanently damaged; whereas the normal heart may vary considerably in its dimensions, and a certain degree of dilatation is extremely common, and is indeed physiological, after severe and protracted exertion. A boy whose heart is in the state just described, the apex-beat diffuse and outside the normal situation, has only to walk smartly across the room two or three times and the apex is back in its place, and its beat is a well-defined push. This is the kind of result which, when duly exaggerated and mapped out in blue and red pencil, we are called upon to admire and wonder at as a miraculous effect of the Schott treatment. Dilatation of the heart as a disease is unknown at the time of life at which youths enter Sandhurst or Woolwich, in the absence of valvular lesion or adherent pericardium, except as a temporary consequence of diphtheria or typhoid fever, or acute rheumatism or other acute disease, or of anaemia or some debilitating influence. When present it can be remedied by fresh air and exercise and favourable hygienic influences, aided when necessary by tonics.

The medical examiners for the army have a very difficult and ungrateful task. It is their duty to pass into the service only such men as they believe to be sound and vigorous, and competent to face the fatigue and hardships of a campaign. They naturally and properly say that, having a superabundance of candidates, the slightest doubt as to a young man's organic soundness is a sufficient ground for his rejection. There will be no difficulty in replacing him by another whose health and vigour are above suspicion; and it must be remembered that a fashionable reproach brought against the examination system for the army is that it places the military virtues of bodily strength and vigour at a discount, and fills the service with weaklings. On the other hand, it is very hard on a young man who has gained a place on the list for admission to Woolwich or Sandhurst, to have the career on which he had set his heart closed to him, to see his hopes and ambitions and aspirations all wrecked. I am quite certain that these considerations are present in the minds of the medical examiners, and that they issue the fiat of disqualification unwillingly. All I

wish to do is to place the results of my experience at their disposal, and also at the service of the anxious general practitioner, who is called upon to tell parents whether their children can be safely allowed to go to school, and to guide them in the choice of a profession for their sons.

Intermittent Albuminuria.—Another source of unnecessary alarm and occasional cause of alleged unfitness for public services is the variety of albuminuria called intermittent or cyclical, or the albuminuria of adolescents. The young medical man on entering practice is apt to look very seriously upon albuminuria or glycosuria or a cardiac murmur; but when one has known cases of albuminuria and glycosuria and valvular disease go on for thirty years, these conditions lose some of their terrors, and experienced life assurance officers and boards do not regard any of these affections as implying the immediate and unconditional refusal of a life. We have to learn to distinguish.

I do not remember to have met with this form of albuminuria in connexion with military cadets, but I have more than once had candidates for the Indian Civil Service and for other branches of the Civil Service who had been refused on account of it. The examination takes place later, and the course of study is more severe. In one of the cases which came before me, the young man had been told to appear again for examination in three or four months and to place himself under the care of his family medical man. He had been assiduously and carefully treated for catarrhal nephritis, had been kept in warm rooms, and put on milk diet and salines. Under this treatment the albumen became more abundant, and anxiety as to his health and life took the place of anxiety with regard to his career. There were just six weeks to the examination when I saw the patient; but in this time by exercise and fresh air, tonics and mild mercurial aperients, the albumen was dismissed.

Intermittent albuminuria does not arise from kidney disease, and it is not a result of mal-assimilation of food, as has been supposed from the fact that the urine passed early in the morning will contain no albumen, while that passed after breakfast does. It is a circulatory phenomenon, and the reason why the urine passed after breakfast contains albumen is, not that the patient has taken food, but that he has assumed the erect position. If he breakfasts in bed the albumen does not appear; if he gets up and moves about, without taking breakfast, it comes. A young American girl I once saw had no albumen in the urine so long as she remained on one floor, but if she went upstairs it was always said to appear.

The pulse in these cases is unstable, at one time manifesting

moderately high tension, at another low in tension. The apex-beat of the heart I have always found to be relatively weak, as compared with the right ventricle impulse, which is fairly strong.

The treatment of intermittent albuminuria should always have for its object improvement in the general tone. An outdoor life, vigorous exercise, a meat diet, such tonics as iron and strychnine, are the measures required, and a single grain of blue pill, or grey powder with rhubarb, or colocynth and hyoscyamus, is usually necessary once or twice a week. Under coddling I have known the coagulated albumen occupy a quarter or a third of the column of urine in a test-tube in a patient who ultimately made a perfect recovery; and one of the most splendid men I ever knew was made a neurotic invalid for life by having been treated for disease of the kidneys when he was merely suffering from intermittent albuminuria.

THE PREVENTION OF CONSUMPTION AND OTHER FORMS OF TUBERCULOSIS

Delivered at the Technical College, Huddersfield, on October 22, 1898
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Gentlemen,—Having been called to Huddersfield on one matter concerning the public health it occurred to me that I might perhaps render a service to my native town and neighbourhood by bringing before you another question of great importance—namely, the prevention of consumption. I claim no special knowledge of the subject and shall have nothing to say which will be new to my medical friends in Huddersfield, so many of whom I see around me to my great satisfaction, and I should like to begin my lecture here by thanking heartily the members of the profession for the cordial support and encouragement they have given me in this matter. You would have learnt from them in course of time how the ravages of tubercle may be stayed, but knowledge percolates slowly, and while it was being brought home to the general public many hundreds of valuable lives might be sacrificed. It is the realization of this fact which has led to the formation of the National Society for the Prevention of Consumption and Other Forms of Tuberculosis.

Prevalence of Consumption.—You are all familiar with the prevalence of consumption. There can scarcely be a person here who has not lost some relative or friend from this disease. Statistics, therefore, are scarcely necessary, but it will perhaps bring more definitely before your minds the serious loss of life to which tuberculous disease gives rise in this neighbourhood if I quote the figures relating to it which are found in the able report of Mr. Annis, your medical officer of health, for the year 1897, and show you on an enlarged scale a diagram which he has made illustrating the proportion of deaths from all causes. You will see what an enormous preponderance consumption claims. The total number of deaths in the borough in 1897 was 1,666, and of these 222, 1 in 7·5, were due to the various forms of tuberculous disease. We are, say, 800 people in this room; 80 or 100 of us will die from consumption or other effect of tuberculosis. In

Great Britain and Ireland there are about 200 deaths each day, and every day more than 200 persons catch this disease. You will all agree that if this terrible sacrifice of life is preventable it ought to be prevented. I can imagine some of you who are cynically minded, and especially such as consider themselves safe from diseases of this kind, saying, "Well, we must die from something; why not from phthisis?" If consumption only weeded out the weaklings there might be something in such a view. But it is often the most gifted, the most useful, the most diligent, the most intelligent, the most amiable who are carried off in this way. Is this not so? And then it is so tedious and trying and often so painful and distressing a way of leaving the world; and so expensive too, frequently exhausting the entire resources of a family.

Infection, not Heredity.—Until the last few years the idea was that consumption was a disease which was in the family, a constitutional tendency which could only be accounted for by heredity and against which it was in vain to strive, an inevitable evil to be submitted to as a decree of inscrutable providence. Added to this was the impression that it was incurable, and thus efforts to prevent and cure it were paralysed. Both these ideas, I am thankful to say, are wrong. Tubercle is not inherent in the constitution. Consumption is not an inevitable disaster, inflicted by a mysterious fate; it is, on the contrary, one of the products of men's ignorance and carelessness. We now know how it is brought about, and it is within the power of man to prevent it. Consumption is "caught," to use a familiar expression, from a pre-existing case; not, let me add at once, communicated directly from person to person by breathing the same air or even sleeping in the same room. There is no need to aggravate the sufferings of the phthisical patient by treating him as a public danger; no need to drive wife or child out of the house to save the rest of the family.

The Bacilli.—The agent in the causation and transmission of the disease is the tubercle bacillus, a minute rod-like organism, which multiplies at an amazing rate. It is visible only under very high powers of the microscope and then only after careful staining. The bacilli obtain access to the lungs or to other organs and if they find the soil suitable, and if they are not destroyed by the defensive agencies which are happily present in the system, they proceed to increase and multiply, compressing and blocking the minute blood-vessels and lymphatics, choking the air passages and air vesicles of the lungs, and forming the little pin-head solid bodies which gave rise to the name tubercle. Eventually the nutrition of the structures is so interfered with that they lose their vitality; inflamma-

tion is set up, and the result is that the tubercles and the portion of tissue invaded by them soften and break down, forming small cavities which extend and coalesce till cavities of considerable size result. This is not the only harm they do, for besides damaging and destroying the tissues they form a poison or toxin which is absorbed into the blood, and by its presence in the circulation gives rise to the fever, sweating, and wasting which attend consumption.

Mode of Dissemination.—We should naturally suppose that these tubercle bacilli being present in diseased lungs by millions would be carried out by the breath, but this is not so. They are not found in the moisture condensed from the breath upon a piece of cold glass or metal, or in fluids through which expired air is made to pass. But they are contained in countless numbers in the expectoration, and become the means by which the disease is disseminated. These microbes are possessed of extraordinary vitality, even outside the body, but there are certain agencies which they cannot resist—heat, sunlight, and fresh air. Fresh air and sunlight are, in fact, the great enemies of all pathogenic organisms. The drying of the expectoration, however, does not kill the tubercle bacillus. Apparently, on the contrary, the desiccated organic matter serves as a protection to the bacilli, so that in the form of dust they survive indefinitely. The expectoration, then, of a consumptive person upon a pocket-handkerchief, or accidentally contaminating articles of dress or the carpets of a room, may sooner or later take the form of dust and so be carried about in the atmosphere. But careless people will spit about in the streets and roads and, when not under observation, in railway carriages and other public conveyances, in public buildings, especially in the corridors and passages, perhaps even in churches or chapels. This is not only nasty but dangerous. There is always the possibility that it may get dried, perhaps on ladies' dresses, which it contaminates, and be dispersed as dust, and it is by the inhalation of germ-laden dust that the seeds of phthisis are planted in the lungs.

We do not realize how ubiquitous dust is. We scarcely know where it can come from when we see it lying on papers, furniture, picture frames, windows, and in the holes and corners into which the diligent housemaid pursues it. You have all seen the motes dancing in the sunbeams crossing the room; you may have seen them in the rays of the lamp this evening. Every mote is a dust particle which may possibly carry on its person numbers of tubercle bacilli. This is how consumption is usually contracted. In every breath we take we inhale an incalculable number of motes or

particles of dust of a more palpable form. Some of these may elude the defences which nature has planted in the respiratory passages, the filtering arrangement in the nose for entangling and arresting microbes, and the cilia of the windpipe and bronchial tubes, and reach the air cells. Then supposing they happen to convey bacilli, if the local and constitutional conditions favour their development the mischief is done. Let one bacillus out of a million find its way to predisposed lungs and consumption will be rife.

Milk.—But there is another way in which tuberculosis is disseminated, and that is by means of milk. Cows are very subject to tuberculosis, and at a certain stage of the disease tubercle bacilli are present in the milk. It is through milk so contaminated that children come to have tabes mesenterica, tuberculous disease of the bowels and mesenteric glands (better known, perhaps, as consumption of the bowels), and, directly or indirectly, tubercular meningitis, or acute hydrocephalus. The diseases of bones and joints to which children are subject are probably also traceable to milk; humpback, hip-joint disease, and the diseases of knees, elbows, etc., which cripple so many children; perhaps so is lupus; and no doubt tubercle is often implanted by milk in early life which develops later into consumption. While blaming the cows we must not forget that a consumptive mother may not only be wasting her own strength by suckling her child, but may be sowing in it the seeds of some of these diseases.

It is interesting to note that asses and goats do not suffer from tuberculosis and to bear in mind that the shrewd physicians of past days used to order asses' and goats' milk for persons threatened with consumption.

You will see how necessary it is that the milk-supply of large towns should be guarded from tuberculous contamination, and I am glad to find that the health authorities of Huddersfield are fully alive to this. The regulations with regard to cowsheds, dairies, and milk-shops on pages 39 to 44 of the medical officer's report for 1897 are admirable. They may be read with advantage by all consumers of milk and by those who keep cows for the supply of milk to their own households. I should like to see the medical officer of health empowered to make an addition, viz., that no person suffering from phthisis should be employed about cows. Consumptive persons should especially be forbidden to engage in milking or in the handling or distributing of milk. I do not like to contemplate the disgusting possibility of contamination of milk by sputa, but we cannot but admit that it might occur.

Meat.—There is no need to consider at any length the contingency of the communication of tuberculous disease by means of meat. This

is adequately provided against here by the public slaughter-house and by the careful inspection of meat. You are all the more secure in Huddersfield in consequence of the excellent relations which exist between your medical officer of health and the butchers of the town. Besides, we do not eat our meat raw, and cooking is fatal to bacilli.

The measures which are required for the prevention of consumption are at once suggested by what I have said as to the methods by which it is disseminated. If we could secure the destruction of all the expectoration of sufferers from phthisis and the freedom of milk from tubercle bacilli we should very soon attain the end of the "National Association for the Prevention of Consumption." This ought to be quite possible, but before entering upon the way in which it can be effected, I must say a few words with regard to the old idea as to family proclivity and hereditary tendency to consumption. This was not a mere theoretical assumption; it was based on observed facts, and nothing could be more foolish than to reject a conclusion founded on experience simply because the explanation was erroneous.

Predisposition.—Although the immediate cause of consumption is the implantation of the tubercle bacillus, it is still perfectly true that consumption runs in certain families and that constitutional predisposition is an important factor in the production of the disease. But it is not actual disease which is transmitted from parent to child, nor will any degree of tendency in the constitution induce it. The hereditary constitutional predisposition is simply a liability to tuberculous disease on exposure to the germs, a vulnerability of the tissues producing a suitability of soil. Two people may be equally exposed to invasion by the bacilli, and one will develop phthisis while the other will not, and we can usually tell beforehand which of the two will succumb and which resist. It may be mentioned that gout seems to reinforce the resistance to infection by tubercle, and, curiously enough, so does the anaemia to which young girls are subject. Besides, however, a constitutional susceptibility, another reason why consumption prevails in certain families is that generation after generation lives under the same unfavourable conditions, as for example in dark, damp, ill-ventilated houses—in houses very often which actually harbour the bacilli left by previous cases.

Preventive measures.—As regards the preventive measures which must be adopted I will first complete what I have already said on this point as to milk. To begin with, an absolute defence against the communication of tubercle bacilli by means of milk is to boil it. But some people, children especially, do not like boiled milk, others

think it does not agree with them. But the desired end, the destruction of the bacilli, can be attained by exposure for a certain time to heat short of the boiling point, which does not affect its flavour—by sterilisation as it is called. This, however, demands a little time and trouble, and in how many households is the simple boiling of the milk too great a trouble? Fortunately, milk is now sterilised on a large scale at many of the more important dairies, and can be supplied in any quantity. As it keeps indefinitely while the bottles are closed it can also be sent to any distance. I tasted some at the Sudbury Dairy on Lord Vernon's estate a few weeks ago which was a month old and as fresh and sweet as on the day it was milked. The railway companies ought to insist on this sterilised milk being kept at refreshment rooms instead of the tumblers of milk which we see exposed on the counter—which may have been standing there for half a day, absorbing bacteria and filthy emanations of every sort. If the railway companies do not insist on it in defence of their passengers I hope the public will do it in self-defence. It is to the defensive instincts of the public that we appeal.

But while we can kill the bacilli in milk we have a right to demand that milk shall be supplied to us which does not contain them. This opens up a large subject. An enormous proportion of the milch cows in this country and over most of the continent is affected with tuberculosis, and they are potential sources of disease. Contrary to one's expectations, tuberculosis is much less prevalent among cows kept for dairy purposes in large towns than in the herds of dairy farms. This is due to the fact that cows which come under the purview of the medical officer of health are inspected and perhaps tested, and such as are diseased are killed off or quickly got rid of in some way. It is a melancholy thing that the greatest danger comes from fresh country milk.

It is with great reluctance that I contemplate the imposition of a new burden on the distressed agriculturist, but pure milk we must have. Fortunately, an easy and certain test for the presence of tuberculous disease in the cow exists in the subcutaneous injection of tuberculin, and already in many herds animals found to be affected are separated from the rest and gradually got rid of. In a few years with precautions against the introduction of new animals affected with tuberculosis the disease can be eradicated. This is a matter for rural and district councils and for landowners. It has, in fact, been taken up by far-sighted and public-spirited landlords, and the Minister of Agriculture, the Right Hon. Walter Long, has given a conditional promise that tuberculin shall be supplied and the veterinary surgeon paid by Government.

To insist on the universal testing of herds and the immediate

slaughter of all affected animals would be ruinous to farmers unless there were compensation, and compensation would involve a demand for money which no Chancellor of the Exchequer could face. It would, besides, be a premium on carelessness and neglect of hygiene. Bad farmers would get rid of sickly beasts of their own making at the public expense. The extinction of tuberculosis in cattle must, I am afraid, be a gradual process, but in proportion as it is accomplished the farmer will be a gainer because his tuberculous cows die early. I have had placed in my hands an appalling list of losses of cows year after year from this disease by an intelligent and industrious farmer. In the meantime we must boil or sterilise our milk, and sterilisation at home is an imperative duty when infants are being brought up by hand. Wholesale sterilisation at the dairy is open to abuse, and there is reason to fear that it may be so carried out as to impair the nutritive value of the milk.

Prevention of the spread of consumption from persons suffering from the disease practically resolves itself into the destruction of the sputa. The sufferers themselves must be brought to understand this, and to realize that unless they adopt and faithfully carry out the necessary precautions they are a standing danger to their family and friends and to the public generally. The families of consumptive individuals must be impressed with the same idea, and the general public must defend itself by insisting on what is necessary for their safety. Convenient receptacles for expectoration have been devised, and one of these patients must carry about with them and conscientiously make use of. Every night and morning, or more frequently, the contents must be destroyed, if possible by burning, and the vessel must be well washed in boiling water and cleansed by some disinfectant, a little of the disinfectant fluid being left in it to receive the expectoration. If no fire is available it is, perhaps, permissible to throw the contents of the spittoon down the water-closet or privy well mixed with a powerful disinfectant, never should they be thrown on an ash heap or out into the street or road or garden or field. Pocket-handkerchiefs should not be employed unless in the form of a square of soft paper, such as is used by the Japanese, which can be at once burnt. Some of my zealous friends advocate notification of consumption, as is done in cases of fever, but, I ask, what are you going to do with the patients when you have notified them? All the hospitals in the kingdom would not hold them, and I doubt whether any effectual inspection and enforcement of precautions would be practicable. The worry would be intolerable. And if we wait till the Legislature enforces compulsory notification years will be lost.

One form of notification, however, is already in force—the death certificate—and action ought to be taken upon this. The room or rooms in which a consumptive patient has lived and died ought immediately to be thoroughly disinfected under the supervision of the medical officer of health. In the case of those who have received public help during life this should be imperative, and others will, I am sure, see the necessity of it in the interest of other members of the family.

Ventilation.—But there are preventive measures to be taken on the part of those as yet in health, and these are of especial importance in the case of those who belong to families in which there is an inherited susceptibility. The most effectual of these is to live, as far as possible, in fresh air day and night, summer and winter. The closely fitting windows to which we attach such importance are a snare and a delusion. When we are sitting in a room with a fire if air cannot make its way in by the window from the open air outside it will come in under the door from the passage and basement. Fear of a draught from a window open at the top leads to that most pernicious of draughts along the floor and about the ankles. Ventilation is of vital importance. But it is necessary by night as well as day. Everybody ought to sleep with the window open and the bedroom ought to be as fresh in the morning as when it is entered at night. I believe—it is a mere conjecture, of course—that if we all slept with open windows the mortality from consumption would be reduced by one-half from this alone. There would be fewer colds and coughs, too.

The Construction of Houses, again, is a matter of great importance. There ought not to be a dark corner in a house, still less a dark passage. Sooner or later a dark passage or staircase will harbour germs, and, as everybody knows, they are the homes of dust. From this point of view some of the extensive flats and gigantic hotels which are now so numerous are a source of danger. In many of them the passages are tortuous and dependent on gas or electricity for light. Efficient ventilation is impossible, and as an enemy of germs even the electric light is a poor substitute for the sun. I used to be greatly astonished to meet with tuberculous disease in families living in splendid country houses, with every advantage of climate, soil, and surroundings, and all the aids to health which wealth and education can command. At last I found that the secret was dark corridors and, in one remarkable case, a dim central hall. I was chatting with an eminent man of science this summer and happened to allude to this conclusion of mine, upon which he told me that he was the sole survivor, I think, of eight brothers and sisters, and that he had

lost almost as many uncles and aunts, all from phthisis, and that the family home was just such a large and imposing but dark and gloomy house as I had described.

I need scarcely mention the necessity of attention to the personal health. Excesses of all kinds diminish the constitutional resistance to tuberculous invasion. Alcohol in excess not only stupefies our brains but the phagocytes, as the cells are called, which attack and destroy microbes.

The Open-air Treatment.—So far I have said nothing of the treatment of those suffering from consumption, and you might be disposed to think that this formed no part of my subject. But it does. Nothing can be more efficacious in preventing the spread of consumption than to cure those already affected by it, unless, indeed, you kill them off out of the way. Now a great deal more can be done to arrest and cure phthisis than was formerly supposed possible, by means of what is called, and what is in truth, the open-air treatment.

We have been in the habit of sending phthisical patients to the shores of the Mediterranean in search of sun, to the mountain snows of Switzerland for a germ-free atmosphere, or on voyages to every part of the world for the healing virtues of sea air, perhaps to special institutions in Germany or the Black Forest. Many returned better, some well, others perished miserably far from home and friends. At length we have become alive to the fact that the curative agent is not any particular air, but simply air. Thus the remedy for consumption lies ready to our hands, and instead of being attainable only by the favoured few is available for all. I hold in my hand a pamphlet by Dr. Philip, of Edinburgh, of which the title is, "On the Universal Applicability of the Open-air Treatment of Pulmonary Tuberculosis," and this is not a mere pious aspiration, but a verified experience. At Edinburgh, on the Norfolk coast, in Ireland, and elsewhere it has been shown that consumption can be treated successfully in practically all parts of our islands. This has revolutionized our ideas and it has created new duties.

Sanatoria.—If consumption is preventable it ought to be prevented—if it is curable it ought to be cured. What is needed for this end is the erection of suitable sanatoria, and these ought to be provided within easy reach of every large town. Certain requirements must be fulfilled: there must be a dry soil, a southern exposure, protection from the east and north, absolute freedom from dust, and, if possible, from fog. The special desiderata are a maximum of sunshine and a minimum of wind and wet. The construction of these sanatoria and their internal arrangements will

be simple. The one thing about them will be that it shall be impossible to close the windows of any room in them day or night. They need, indeed, be little more than sleeping-sheds ; but of course they would be made as cheerful and bright as possible, though pictures are almost inadmissible, as the frames harbour dust.

The patient will spend the entire day in the open air, wet or fine, warm or cold. Movable shelters will be provided to shield him from cold winds and to protect him from rain. On no pretext will he sit indoors. The amount and kind of exercise he may take will be determined by his temperature and strength, and his life will be regulated in every respect. No great barrack-like building will be required. A sanatorium will usually consist of a number of small separate pavilions, which may be erected one after the other, and I may venture to point out the opportunity to any one who desires to be a benefactor to Huddersfield of building one of these pavilions by way of a beginning.

An idea which finds favour is that at first these sanatoria should be self-supporting—should, in fact, be for paying patients. Some part of the disease could thus be dealt with before funds could be raised to provide for consumption among the poor. The association which I have the honour to represent as chairman of the organizing committee has for one of its objects the promotion of the erection of sanatoria in all parts of the country. It is to be hoped, and indeed confidently expected, that local branches will be formed for the purpose of ministering to local needs. Such branches will collect and expend their own funds, and will erect sanatoria at any suitable spot in the neighbourhood so that the patients will not have to go far from home. The special function of the National Association will be to co-ordinate the work of different branches, to stimulate public interest in the prevention of consumption, and to co-operate with all the agencies which have for their object the promotion of public health.

We shall bring before county and district councils the question of milk, and aid health authorities to secure the construction of houses which shall not be standing invitations to disease, and the destruction and reconstruction of such as are, and to promote ventilation and cleanliness. Clergymen and ministers of religion, who see in the way of duty so much of the suffering and distress occasioned by consumption, will, we think, eagerly grasp the opportunity of informing themselves how the disease may be combated and prevented and of disseminating the information among families, thus reinforcing the efforts of the medical man. Schoolmasters and mistresses, again, can do much to teach children the lessons of health and self-preservation. I have only time to hint at a very

few of the ways in which a great national association may bring about the prevention of a terrible national loss and the removal of a dark blot on the national good name; and it is because more can be done by a general, simultaneous, and united effort than by any number of isolated local efforts that I give this lecture here to-day. The health authorities of the town have done me the great honour to invite me here to open formally a splendid fever sanatorium. I hope in a year or two to come down again to take part in the opening of a sanatorium for the open-air treatment of consumption, and I hope that in the not remote future, when physicians are lecturing upon tuberculous affections, it will be on examples looked upon as rare and extraordinary cases and not, as at present, furnishing a very large part of their work.

NEUROLOGY

AN ATTEMPT TO REMOVE THE DIFFICULTIES ATTENDING THE APPLICATION OF DR. CARPENTER'S THEORY OF THE FUNCTION OF THE SENSORI-MOTOR GANGLIA TO THE COMMON FORM OF HEMIPLEGIA. (Broadbent's Hypothesis of the Bilateral Association of Nerve Nuclei.—Ed.)

British and Foreign Medico-Chirurgical Review, 1866

THIS theory, as is well known, is, that the thalamus is the organ of conscious sensibility, to which all impressions made on peripheral sensory nerve-fibres must be transmitted in order to be recognized as sensations, and the corpus striatum the organ or instrument of voluntary motion—the downward starting point of volitional motor impulses, or it might be said of all cerebral motor impulses. These two ganglia are again associated according to the theory of Dr. Carpenter in sensori-motor action, impressions reaching the thalamus being passed on to the corpus striatum, and giving rise to automatic movements differing from those which have their centre in the cord, only in being accompanied by sensation.

The common form of hemiplegia is caused by haemorrhage, or softening in one or both of these bodies on one side, and the difficulties presented are : that the thalamus and corpus striatum being considered respectively the organ of sensation and motion of the opposite half of the body, the motor paralysis is not general in that half, but affects only the limbs, tongue, and face, while sensation either escapes altogether or is only partially lost. These difficulties have hitherto prevented the theory from obtaining general acceptance.

As different interpretations of the results of experimental and microscopic investigations of the spinal cord and medulla oblongata have been given by different physiologists, it is necessary to state briefly the views I hold as to the function and mechanism of these parts.

The grey matter of the cord then I look upon as containing a series of sensory and motor nerve-nuclei connected together transversely, and fused longitudinally into a continuous chain. With

the posterior and anterior nerve-roots they constitute an apparatus for automatic or reflex action. From the sensory nerve-nuclei communications pass upwards in the grey matter to the thalamus, these crossing in the cord; to the motor nuclei, fibres descend in the antero-lateral white columns from the corpus striatum, decussating at the point of the medulla. The posterior white columns are longitudinal commissures between the superimposed nuclei for co-ordinated movements.

The medulla oblongata is simply a more highly specialised portion of the cord, but has in addition to sensory and motor nerve-nuclei certain accessory ganglia. The constituents are also rearranged. The nerves given off from it having special local distribution and functions of great importance, the nuclei are large, and distinct one from another. The reflex actions also which have their centres here, involve the co-ordinated action of an extraordinary number of muscles, and are of the highest importance to life; the provision for this co-operation by means of commissural fibres must be of corresponding extent. But this localization, or individualization of nerve-nuclei, and linking together of sensory and motor nuclei confers upon them no new property. Voluntary motion does not originate in a motor nerve-nucleus of the medulla, and sensation is not recognized as such by a sensory nerve-nucleus. Each is connected with the corresponding sensory or motor ganglion, thalamus or corpus striatum, just as are the nerve-nuclei of the cord, the communicating fibres crossing the septum, and ascending in the opposite half of the medulla and pons.

These remarks may also be applied to the pons, so far as concerns nerve-nuclei and the longitudinal tracts of fibres.

The thalamus being thus looked upon as the seat of sensation, and the corpus striatum as the instrument of volitional (ideational) action; the pons, medulla, and cord, being considered merely as subsidiary mechanism, the questions arise in hemiplegia, caused by injury to one or both of these bodies:—

1. Why is not sensation more frequently and profoundly affected?

2. Why is not the entire half of the body, head, and neck paralysed as to voluntary motion, instead of merely the limbs, and in a partial degree the face and tongue?

Affection of Sensation in Hemiplegia.—The first of these questions has recently been put forward prominently by Dr. Hughlings Jackson, in vol. ii. of the *London Hospital Reports*, as one of the discrepancies between what he terms "medical" physiology and "school" physiology. He exaggerates the discrepancy, however, for he says, "As a rule, there is no loss of sensation any-

where." In opposition to this assertion, I might quote any number of authorities, and I can state from my own observation that sensation is very frequently diminished, sometimes very greatly. I have tested it by pricking and pinching, by the compasses, and occasionally by hot substances, the sound side being always used as a point of comparison. This does away with the idea that a subjective complaint of "numbness" has been set down as a loss of sensation. Moreover, it is not the sensibility of the limbs merely, where the motor paralysis is most marked, which is affected, but also of the face, chest, and abdomen.[1]

It is a fact, however, that in common hemiplegia sensation is never (so far as I know) totally lost, and that it is often altogether unaffected when the motor paralysis is complete, and this requires explanation. First, how is it that sensation so frequently escapes when motor power is lost, while the converse never occurs?

A reason for this, almost in itself sufficient, is found in the relative situation of the two bodies. The corpus striatum, the motor ganglion, is in front of, and external to the thalamus, and may be extensively damaged without involving the thalamus or the fibres passing from it to the cord. The thalamus, on the other hand, lying behind the corpus striatum, and upon the fibres connecting it with the cord, can scarcely be seriously affected without injury to these fibres or the corpus striatum itself.

Again, the thalamus, according to hypothesis, standing with respect to the corpus striatum in the relation of a sensory to a motor nerve-nucleus, it would almost follow that severe injury to the former would paralyse the latter by inhibitory influence, even when the injury was confined to the thalamus, and did not reach the corpus striatum directly or indirectly.

It still remains to be explained, however, why the loss of sensation is not as complete in degree when the thalamus is the seat of softening or haemorrhage, as the loss of motor power in the limbs when the corpus striatum is affected.

A parallel is furnished by disease of the spinal cord, motion being almost always first and most profoundly affected, and both are explained by Dr. Brown-Séquard's experiments on the cord. He found a remarkable difference in the results of section of the white motor columns, and of the grey matter along which the sensory impressions travel. The cutting across of a group of fibres in the motor tract was followed by a certain appreciable muscular paralysis, but considerable injury might be done to the grey matter

[1] I ought to add that my observations have mostly been made on recent cases, Dr. Jackson's on old cases, and that sensation is recovered more rapidly than motion.

before any loss of sensation became apparent, and while a single slender bridge of grey substance remained a considerable degree of sensibility persisted in the whole of that part of the body behind the seat of the injury. The entire sensory tract resembles in structure this grey matter of the cord, and the thalamus itself, instead of presenting like the corpus striatum distinct grey matter with white fibres plunging into it, consists of an intimate admixture of cells and fibres.

Without pretending to explain this diffused transmission of sensory impressions along the cord, we may fairly suppose it to prevail in the higher part of the sensory tract, and to be shared by the thalamus. If this be admitted, it is clear that only such an amount of destructive change, as should leave no fragment of this body in relation with the sensory tract, would produce complete anaesthesia. We should, in fact, expect that injury to the thalamus would manifest itself rather in inhibitory paralysis of the corpus striatum than in marked loss of sensibility.

I have left out of this consideration the special senses, recognizing, however, that their exemption constitutes a grave difficulty. If their nuclei, say of the gustatory or auditory, are centres of sensation independently of the thalamus, there is no reason why that of the trigeminus should not have the same property, which would be fatal to the theory of the office of the thalamus. If, on the other hand, these nuclei are supposed to stand in the same relation to the thalamus as the sensory nuclei of the cord, the exemption of the special senses requires special explanation. This, I believe, may be given, but it will better form the subject of a separate communication, and the difficulty must be left where it stands.

Many physiologists locate sensation in the medulla or pons. Some even suppose, that all the sensory nerve-nuclei of the cord are centres of sensation. These hypotheses would explain the absence of any impairment of sensibility in hemiplegia, but the loss of sensation often met with would furnish a greater difficulty than the one escaped from. I cannot now go into the objections which might be urged against these views, but pass on to the question respecting motor paralysis.

Motor Paralysis in Hemiplegia.—The difficulty here, as has before been stated, is, that the limbs, and in less degree the face and tongue, only are paralysed, and not the entire lateral half of the body.

In attempting an explanation of this, it becomes necessary to specify the muscles paralysed, and more particularly to determine the precise character of the facial paralysis in hemiplegia. The contrast between facial paralysis proper and facial hemiplegia has

been frequently described. Dr. Todd accounted for the differences observed in the two instances, by supposing that in hemiplegia the motor division of the fifth was affected, the seventh escaping, but this view, I suppose, is not now held by any one who has given attention to diseases of the nervous system. The distortion of facial hemiplegia, as in facial paralysis, is generally recognized as being due to paralysis of the seventh nerve, but as to the exact character and extent of this paralysis there is still diversity of opinion. Dr. Hughlings Jackson, in the paper before referred to, says, " the paralysis is simply of a small part of the face near the angle of the mouth—not of the portio dura, but of part of it." It is true that the paralysis is most evident in the part of the face referred to, but no muscle supplied by the portio dura altogether escapes paralysis, while, on the other hand, the paralysis is complete in none of them.

It is not necessary to describe the expressionless condition of the affected side of the face, or the partial obliteration of the muscular markings; but it is important to observe that this extends in a slight degree to the forehead, where the wrinkles in a recent and severe case when well marked will be found slightly smoothed out, and the vertical furrow produced by the corrugator supercilii a little less deep. The orbicularis oculi which furnishes the most striking point of contrast between " facial paralysis " and " facial hemiplegia," itself gives evidence of impaired power. For a short time after a severe attack, it is obviously weakened, and this may be rendered more evident by bidding the patient close both eyes powerfully, or again, by asking him to wink the eyes alternately. The eye on the hemiplegic side cannot be closed alone, and the voluntary contraction of the orbicularis of this side will be seen to be less forcible. On the other hand, it is equally important to note that in the region about the angle of the mouth where the paralysis is most marked it is not complete. The dragging over of the mouth to the sound side is not so great as in paralysis of the portio dura, and imperfect movements may be observed in the paralysed muscles.

All the facial muscles, therefore, are partially paralysed, though in very different degrees, none completely so. I should place them in the following order. Least of all the small muscles of the alae nasi, the orbicularis oculi, the occipito-frontalis, and corrugator supercilii; next, at a long interval, the orbicularis oris; most of all the straight muscles going to the lips and angle of the mouth. That the orbicularis oris is much less affected than these last mentioned, is evident from the perfect closure of the mouth which can be effected. From this fact also,

the attempt to whistle, or blow, sometimes directed for the purpose of showing the paralysis, rather diminishes than increases the facial distortion.

The muscles supplied by the portio dura, however, are not the only ones in the face which give evidence of paralysis. The masticatory muscles, though they take no part in producing the distortion of the features, do not altogether escape. If the patient is told to close the jaw firmly, the masseter and temporal of the sound side may be felt to come first into action, and to contract the more powerfully; sometimes those of the affected side only act after two or three attempts.

In the tongue, as in the face, there is evident, but incomplete paralysis, the only manifestation of it being deviation when it is protruded, usually towards the paralysed side.

The abdominal muscles may be made to show a certain degree of paralysis much in the same way as the masticatory muscles. In respiration they act on the two sides with perfect equality, these movements being automatic; but in other actions it may be seen that the muscles of the affected side are weakened. Thus, though a hemiplegic patient is able to raise himself from the recumbent to the sitting posture with little or no help from his sound arm, by means of the recti abdominales, and the hand placed upon the abdomen feels the muscles of both sides contracting powerfully, still as in the case of the masseters, those of the affected side are somewhat later than the others, and do not act with the same energy.

The limbs are the only parts in which motion is altogether lost. In a severe case, I have found not only every muscle of the arm paralysed, but the scapula perfectly motionless. The pectorals, the latissimus dorsi, and trapezius, the latter in movements of the head also, were flaccid, and apparently the more deeply seated muscles, the serratus, rhomboidei and levator, were equally paralysed. In the leg, the loss of motor power is rarely so complete as in the arm.

These are the muscles paralysed. It is, however, even more necessary to specify the muscles which escape paralysis, since it is this exemption and not the fact of paralysis which requires explanation. The muscles incompletely paralysed also, will again have to be alluded to, the partial exemption having to be accounted for as well as the complete escape.

The muscles, then, which may give no indication whatever of paralysis are those of the eye, neck, back, and chest. The movements of the two eyes are as perfect as ever; there is no paralysis, therefore, of the third, fourth, or sixth nerves of the affected side.

As to the neck, it is sometimes stated that the sterno-mastoid is partially paralysed, but I have never observed this, and in cases where the use of the arm and leg has been entirely lost, I have found the rotatory and the backward and forward movements of the head to be executed as well as ever. Inclination of the head towards one or other shoulder does not seem always as easy as usual, but it cannot be said that any muscles of the neck give appreciable indications of paralysis. No difference again can be detected in the movements of the two sides of the chest, or so far as I have been able to make out in the back. There are other sets of muscles which, without showing any obvious paralysis may be made to furnish evidence of impairment of independent volitional action. These are the levator and orbicularis of the eyelid, the masticatory, and abdominal muscles, as already described. Others, again, are manifestly paralysed, but not completely, as the facial and lingual muscles.

In all these instances, it is the persistence of purely volitional motor power which is spoken of, and not automatic action of any kind. The reflex respiratory movements of the chest and abdomen of course go on, but there remains also the power of taking a deep inspiration, or making a forcible expiration at will, which is not reflex but voluntary. Again, there is the act of rising into the sitting posture executed by the abdominal muscles. So also in the case of the eyelids, in addition to the automatic winking which is not arrested as it is in paralysis of the portio dura, there is the voluntary closure of the lids, more or less firmly. The movements of the eyes have been spoken of as "sensori-motor," but it is evident that they are only so in the same sense as all motion is influenced by a guiding sensation, and are as strictly voluntary as any movements in the body.

If then, the corpus striatum is to be regarded as the centre of all volitional motor impulses, these exemptions, partial and complete alike, require explanation. So far as I know this has only been attempted in the case of the facial and thoracico-abdominal muscles, and the explanation has usually turned on the confusion of automatic with voluntary movements.

Former Attempted Explanations.—With respect to the thoracico-abdominal muscles, the explanation usually given is somewhat as follows: "That their habitual and constant action being in the reflex respiratory movements, they are to some extent withdrawn from the influence of the will, and are consequently not affected when voluntary motor power is lost." This which has a very plausible sound will be seen on examination to be absurd. It is in effect, equivalent to saying, that because these muscles are

comparatively seldom called into action by the will, it is reasonable to expect that they will still be reached by volitional impulses when other muscles more constantly acted upon by the will are completely cut off from its influence. The persistence of the reflex respiratory movements is easily understood—the mechanism on which they depend is not damaged, but this does not explain the voluntary action still found possible in the muscles of the affected side.

Van der Kolk employs a similar process of reasoning in attempting to explain the exemption of these muscles, by reference to the relation of the lateral columns of the cord with the nucleus of the vagus. These columns have been shown by the experiments of Schiff, to serve for the motions of the trunk, and therefore for the respiratory movements. Van der Kolk finds that their fibres terminate for the most part in the nucleus of the vagus, this connexion being part of the respiratory apparatus. He concludes, further, that in consequence of this relation, the function of the lateral columns does not depend directly on the will, though to a certain extent influenced by it, but that they are brought into action specially by a stimulus from the vagus. That is, because these columns form part of an automatic apparatus of which the nucleus of the vagus is the centre, when they are called into action by a totally distinct power, volitional, this also has its seat in this nucleus. His own researches have shown this idea to be untenable. Fibres are found to pass upwards from the vagus, through which the will is supposed to influence the respiratory movements, and *a fortiori*, these, or an independent set of fibres, must be required for such actions as sitting up. When these fibres are cut across, as they must be in hemiplegia, if they are connected with the corpus striatum, voluntary control over the trunk muscles ought to be lost, whether they pass directly into the lateral column or influence it indirectly through the nucleus of the vagus. The persistence of volitional motor power in the thoracico-abdominal muscles in hemiplegia, therefore, is not accounted for by the relation between the lateral columns of the cord and the nuclei of the pneumogastrics.

The most recent attempt to account for the partial character of the facial paralysis in hemiplegia, is by Dr. Saunders, in a paper in the *Lancet* for 1865, vol. ii. p. 478.

After showing that it is the seventh nerve which is paralysed, and not the fifth, as was stated by Dr. Todd, he points out that the facial muscles have three distinct modes of action, as respiratory muscles, reflex; as muscles of expression, emotional; and as voluntary muscles, strictly speaking. He supposes that for each of these different kinds of action, the trunk of the portio dura

contains a distinct set of fibres connected at its origin with different excitor centres, volitional, emotional, and reflex respectively, and that destruction of the volitional centre, or of the fibres leading to it, may leave untouched the emotional and reflex centres and communications, permitting the muscles to be called into action through these. Even, if this hypothesis be accepted, it affords no explanation. According to it, purely volitional power should be completely lost, but this is not the case as has been pointed out. A considerable degree of it remains, even in muscles supplied by the portio dura, and, as has before been said, persistence of automatic action does not explain the possession of voluntary control.

In order to be more explicit, and render the insufficiency of this explanation more clear, the orbicularis oculi may be taken as an illustration. The habitual winking movements of the eyelids are automatic—reflex, as they would be termed by some, or sensori-motor, according to others. But we have also the power of keeping the eyes shut at will for any length of time, and of closing them with varying degrees of force, these being distinct exercises of voluntary power. Applying Dr. Saunders' explanation, some of the nerve-fibres supplying this muscle will be connected centrally with the nucleus of the fifth nerve for the automatic action; others pass to the corpus striatum, and convey the volitional influence. These latter being cut across on one side in hemiplegia, the winking should go on as usual, but the power of forcibly closing the eye of the affected side should be lost. This is not the case, a certain degree of weakness may be apparent, but both eyelids can still be closed at will, and held down with considerable force.

Dr. Hughlings Jackson also, as I gather from a note to the article before referred to, supposes the portio dura to break up within the medulla, and to proceed to different parts of the nervous centres, not, however, precisely on the same grounds as Dr. Saunders, but on account of the wide distribution and varied functions of the nerve. If this were so, it would still fail to account for the persistence of voluntary motion in any of the muscles supplied by it, when the centre of volitional action was destroyed.

But it is an ascertained fact, that the facial nerve does not split up in the way here supposed, and the varied actions, automatic, emotional, and volitional, can be explained without any such hypothesis. The nerve passes entire to its nucleus, and it is by the communications of this nucleus with the different excitor centres, that the various kinds of movement are brought about, with the nucleus of the vagus for respiratory movements, with the fifth for sensori-motor, and with the corpus striatum for emotional and

volitional actions. The same fibres in the nerve-trunk convey the impulse from the nucleus to the muscles, from whatever centre the impulse may originally have been derived.

Dr. Jackson has again pointed out the interesting fact, that the muscles of the sense-apparatus escape as well as the special senses themselves, but they are not the only ones exempt, and it is not pretended that any satisfactory explanation is thus obtained.

The Probable Explanation.—We come back then to this point, that if the corpus striatum is to be considered the organ of volitional action, an explanation is still required of the incomplete paralysis of the opposite side of the body, neck, and face, when this body is the seat of disease.

The key to this, I believe, is to be found in a comparison of the muscles paralysed with those exempt from paralysis, as to their habitual action. A striking difference is at once noted. Thus the arms (in which the paralysis is complete), are entirely independent in their movements, the one of the other, are altogether dissociated in their action, and habitually engaged in totally different motions. The muscles of the trunk, on the other hand (which escape paralysis), act in pairs, are almost always bilaterally combined in their action, and the two sides engaged in similar and associated movements. We move one arm, or one leg, while the other is quiet, or executing a totally different action. We find it impossible to expand one side of the chest without the other, or to move one eye without the other, and extremely difficult to throw into action the muscles of one side of the abdomen without the other, impossible, indeed, to do this forcibly.

The parts paralysed then, are such as have the power of acting independently of the corresponding part of the opposite side. The muscles which escape, are those which act only bilaterally, or in concert with the corresponding muscles of the opposite side.

But when muscles habitually act together, and rarely or never independently of each other, the nuclei of their nerves are usually connected by commissures.

The hypothesis suggested by these considerations is, *That where the muscles of the corresponding parts on opposite sides of the body constantly act in concert, and act independently, either not at all, or with difficulty, the nerve-nuclei of these muscles are so connected by commissural fibres as to be* pro tanto *a single nucleus. This combined nucleus will have a set of fibres from each corpus striatum, and will usually be called into action by both, but it will be capable of being excited by either singly, more or less completely according as the commissural connexion between the two halves is more or less perfect.*

The existence of this transverse commissural communication between corresponding nuclei is not hypothetical, the fibres have been observed and described, and the association effected by them is considered necessary to harmonious bilateral action, but so far as I know the use here attributed to them, that is of conveying to one an impulse received by the other, has not been suggested.

According to this hypothesis then, if the centre of volitional action of one side is destroyed, or one channel of motor power is cut across, the other will transmit an impulse to the common centre, and this will be communicated to the nerves of the two sides, equally, if the fusion of the two nuclei is complete, and there will be no paralysis—more or less imperfectly to the nerve of the affected side, if the transverse communication between it and its fellow is not so perfect, in which case there will be a corresponding degree of paralysis.

This will be better understood when illustrated by examples. The nuclei of the two third nerves, for instance, are situated close to the median line, high up in the floor of the fourth ventricle, and are so intimately connected together that they may be considered as one single centre, each half of which receives fibres from the corpus striatum of the opposite side. Supposing now the right corpus striatum be injured, voluntary impulses from the left will pass to the right nucleus only, but the two nuclei being fused into a single centre, this is called into action equally throughout, and the muscles of the left eye act as perfectly as those of the right. In the case of the portio dura, on the other hand, the communication between the nuclei is imperfect. Here, then, the same injury having occurred, the left nucleus receiving no impulse from its own motor ganglion, receives only an imperfect impulse through the partial communication between it and its fellow supplied by the uninjured corpus striatum, and the muscles supplied by it are partially paralysed.

Applying now the hypothesis generally, it ought to be found, first, that the paralysis in any given set of muscles is exactly proportionate to the individuality of their action, and their independence of muscles of the other half of the body.

And secondly, when any set of muscles having a certain degree of independent action partially escapes paralysis through association with muscles of the opposite side, that associated movements only are possible on the affected side, and not independent unilateral action of these muscles.

I think it may fairly be said, conversely, that if these tests are complied with, they furnish strong evidence of the truth of the hypo-

thesis. Taking then the different parts of the body in succession, and employing these tests, we find, as I have before stated, that the limbs which alone have perfect unilateral independence of action, are alone liable to complete paralysis in the common form of hemiplegia.

On the other hand, the ocular muscles are never paralysed, and these have no unilateral independence. The two eyes always move together. It is impossible to move one and keep the other fixed, or to turn one up and the other down. Individuals are occasionally met with who can at will exhibit a convergent squint, but this apparent exception to the rule is really only an exaggeration of an associated movement. The centres from which the motor nerves of the ocular muscles of the two sides proceed are inseparably joined, and if they act at all must act together. We thus also see why strabismus in cerebral affections implies something more than disease of the central ganglia.

In the case of the thoracic muscles, again, we have the same point illustrated; no unilateral independence of action; no unilateral paralysis. Between these extreme cases of complete paralysis and perfect exemption, we have the instances of partial paralysis already enumerated, which afford opportunities for the application of both tests.

The eyelids habitually act together both in the habitual automatic winking, and in the strictly voluntary movements already mentioned, but independent action of one without the other is not impossible. The power of winking one eye is, however, an educational acquirement, some learn it easily, others with difficulty, others again never master it, and it is not uncommon to meet with persons who can wink one eye alone, but not the other. In hemiplegia accordingly, paralysis is not very apparent in the eyelid. For a time after the attack it is weakened on the affected side, but to render this evident it is often necessary to tell the patient to close the eyes firmly. In accordance therefore with the first test, we have, with little unilateral independence, slight paralysis.

The second test is applied by bidding the patient close each eye alternately. He will be unable to do this on the paralysed side, and in making the attempt it will usually be seen that the orbicularis of the sound side contracts, against the wish of the patient, while that of the affected side remains comparatively passive.

A comparison between the orbicularis oris and the straight muscles, levator, zygomatic, buccinator, etc., again is an illustration of the fact, that the paralysis is proportionate to the independence. It is very easy to draw one angle of the mouth in any direction, the other remaining in position—difficult to compress the lips

firmly on one side only. Accordingly, as has been stated, the orbicularis is paralysed to a less degree than the straight muscles.

As to the remaining muscles supplied by the portio dura, in those of the forehead and brow, and of the ala nasi, there is little independence of action and little paralysis. In those of the cheek and lips we have increased independence and marked paralysis.

The movements of these parts are usually symmetrical, and a certain degree of association of the nuclei is presumed to exist, the paralysis consequently is not absolute; but the two sides move independently of each other with perfect ease from the earliest period of life, this association of the nuclei therefore is only partial, and not sufficient to prevent distortion of the features.

The second test does not give such decided results as in the case of the eyelids. While the paralysis is such as to give rise to marked facial distortion there is no power of independent motion in the affected side; but this soon returns, and may be observed when the eye of that side cannot be closed alone. This may be to some extent due to the power of fixing the muscles on the healthy side.

I have already described the very slight indications of paralysis which may be obtained from the masticatory muscles. This is in exact proportion to the small degree of independent action they possess. The muscles of the two sides always act in concert, and any one making the experiment will find it impossible to exercise any considerable force by the masseter of one side without bringing into action that of the other, even if a hard body be placed between the teeth on the side attempting to act alone.

Again, in the abdominal muscles, the degree of paralysis and the power of unilateral action correspond very nearly with what has been stated of the muscles of mastication. It will be found almost impossible to throw the muscles generally of one side into powerful action without those of the other side. In inclining the body to one side or the other, the rectus abdominis will act without its fellow, and it is in similar movements that impaired power may be shown in hemiplegia.

The tongue furnishes a very interesting illustration of paralysis of unilateral action and exemption of bilaterally combined movements. This organ is usually perfectly symmetrical as to its lateral halves. They are elongated, shortened, flattened, or thickened together; the only unilateral movements are those in which the tongue is carried bodily from side to side and the tip pointed right or left, or in which one margin is depressed and the other raised. The apparent exception to this rule, when one edge of the tongue is rolled up, is produced by pressure of that side against the teeth, and is a result of the lateral motion mentioned. Accordingly, in

hemiplegia, there is no loss of symmetry in the tongue; it is still flattened and elongated as a whole; there is no impairment of motility in one half, but there is deviation of the tip when it is protruded.

The complicated arrangement of the muscles of the back makes it difficult to apply the tests to them. It is, however, certain that they give little evidence of paralysis. Many of their actions are automatic balancing movements, and the two sides almost always act in concert. Even when the body is inclined to one side, the muscles of one side are contracting, to regulate the movement and prevent falling over, while those of the other are employed in curving the spine. In putting a hemiplegic patient through these movements, he readily loses his balance, and it is difficult to make out any difference between the two sides. In attempting to raise the hip he leans over to the opposite side.

The only real difficulty is met with in the neck. As has been already stated, the head is rotated from side to side, or inclined forwards or backwards as readily in a patient with hemiplegia as in a sound man. The inclination towards one or other shoulder does not seem quite so easy or so perfect. The forward and backward movements would be accounted for by the bilateral action of the muscles engaged, the recti antici, and the muscles of the back of the neck. The rotatory movements do not admit of so easy an explanation.

But it is to be noted, that although in rotation no two corresponding muscles are engaged, yet there are associated in it muscles belonging to opposite sides of the body. Thus, in turning the head towards the right shoulder, the left sterno-mastoid anteriorly, and the right inferior oblique atlo-axoid posteriorly, are brought into action, and vice versa. Looking now at the nervous supply of these muscles, it will be seen that it is quite possible for their nuclei to be associated, and indeed very probable that they are, the sterno-mastoid receiving a branch from the spinal accessory, some of the twigs of which come off from the cord at the same level as the second cervical, which supplies the inferior oblique. According to this, then, the left sterno-mastoid in hemiplegia of that side will receive an indirect impulse through the nucleus of the second right cervical, and so escape paralysis. The association of nerves of opposite sides is not without parallel; as, for example, in the instance of the third and sixth; and the supposition of a relation between the nuclei of the second cervical and spinal accessory would furnish an additional reason for the scattered origin of the latter.

As to the lateral movements of the neck they resemble the

corresponding movements in the back, both sides acting, one limiting, the other effecting the motion.

I think I may say, that the facts here given furnish a considerable balance of evidence in favour of the hypothesis I have advanced. Its simplicity and its general applicability are of themselves recommendations, and I venture to hope that further observation will clear away the few difficulties which remain, and place it, and with it Dr. Carpenter's theory of the function of the corpus striatum, on a firm basis.

Some interesting corollaries might be pointed out, and the association of the nerve-nuclei will be found to bear on other cases than those of hemiplegia, but any discussion of these would be premature.

ON A CASE OF RIGHT HEMIPLEGIA, WITH DEVIATION OF THE EYES TO THE LEFT, AND APHASIA

The Lancet, May 5, 1866

CASES which are mutually illustrative, or which present some remarkable prominent symptom not fully investigated, become much more valuable when a series of them can be brought together than while they are isolated and scattered at long intervals in different records. Attention has recently been drawn to deviation of the eyes in hemiplegia in the case of Dr. Whewell, so ably related by Dr. Humphry, and by the note of Dr. Hughlings Jackson; and Dr. Russell Reynolds has, in *The Lancet* of April 21, recorded another most interesting case of the kind. To these I seek to add a third, now under observation at St. Mary's Hospital, in which another condition exists, which, in the hands of Dr. Jackson, has been the subject of some of the most interesting and valuable papers that have recently been written on affections of the nervous system —loss of the faculty of language.

(After a history of the case and an account of the onset, he says):—

On March 31, there was paralysis, with rigidity, of the right arm and leg, and of the face on the same side; also deviation of the tongue to the right, and great impairment of speech.

When seen by me on April 2 his head was turned to the left; there was complete loss of power in the right arm and leg; no notable rigidity. The mouth was drawn slightly to the left, and the right side of the face was comparatively expressionless. The *eyes were strongly deviated to the left*; their axes parallel; the right pupil somewhat the larger. When made to look to the right the entire head was turned, and the eyes rarely reached, never passed, the middle line. Right conjunctiva suffused.

(He then describes the aphasia and gives details of the illness and continues as follows):—

April 21. The temperature has been observed and found to be uniformly higher in the right than in the left axilla, 98° F. and

97° F. respectively. His general condition is improving, his appetite good, and there are no symptoms which immediately threaten life. The deviation of the eyes, as has been seen, is diminishing; the aphasia has become more complete.

It may perhaps seem premature to attempt any explanation of the phenomenon in question at present. One point to be ascertained is its frequency. It certainly does not occur in every case of hemiplegia, and I am disposed to think is not common. Until I looked over my notes I thought this the first instance in which I had observed it, but I find it noted in a case watched by me in 1859.

Putting together the facts respecting the symptom itself, we find that the eyes are always turned towards the sound side; that the condition is temporary, and may be looked upon, therefore, as a phenomenon of attack. It cannot be called transient, lasting, as it has done in the present case, twenty-one days. Another fact, which may or may not have value, according as it may be found to occur constantly or not, is, that in Dr. Russell Reynolds' patient, and in both mine, there was temporary rigidity of the paralysed limbs. In the case of Dr. Whewell no mention is made of rigidity, but the paralysis was very slight.

It is yet merely matter of conjecture whether the lateral deviation of the eyes is in any way due to the situation of the haemorrhage. In the case of Dr. Whewell the clot was found in the lower, anterior, and outer part of the corpus striatum, which, by the way, accounts for the slight paralysis. The aphasia in the case now related points to lesion in the same situation. If Dr. Reynolds' silence on the subject of sensation may be interpreted to mean that this was not affected, this would lead us to suppose that the final attack of haemorrhage was at any rate anterior to the thalamus.

Dr. Hughlings Jackson has mentioned a paper of mine, which will be found in the April number of the *British and Foreign Medico-Chirurgical Review*; and as the hypothesis I there advance receives strong confirmation from the phenomenon under consideration, and offers some explanation of it, I may be permitted to refer to it. In the common form of hemiplegia, we find that, while the arm and leg may be completely paralysed, the face and tongue are only partially affected, and the ocular muscles, the orbicularis oculi, the thoracic muscles, diaphragm, and others, completely escape. I account for this, which had not previously been satisfactorily explained, by the bilateral association of the nerve-nuclei of the exempted muscles. It will be observed that those muscles which escape are such as usually act in concert with corresponding muscles of the opposite half of the body, and with difficulty, or not

at all, independently of them; and that those which are partially paralysed are such as usually act symmetrically with their fellows of the opposite side, but can readily be called into action alone; while those completely paralysed are such as possess entirely independent unilateral action. This at once suggests the explanation. The nerve-nuclei of muscles which always act together—say of the two superior or inferior recti of the eye—are connected by commissural fibres, so as to be to all intents and purposes a single nucleus; and since there is no unilateral independence of action, there can be no unilateral paralysis, any stimulus the nucleus of the sound side may receive being at once and equally communicated to the other. Partial paralysis is explained by the commissural communication being imperfect when a certain degree of unilateral independence exists.

To apply this hypothesis of association of nerve-nuclei to the case of lateral deviation of the eyes. This, as is at once evident, is due to paralysis of the abducens on the paralysed side, and of the adducens on the sound side. These muscles always acting together, the nucleus of the sixth nerve and of that part of the opposite third which supplies the internal rectus, are associated so as to constitute in effect a single nucleus (whether it is, as Van der Kolk supposes, that the fibres of the sixth can be traced to the nucleus of the third or not, is of no great consequence). Here, however, instead of the sound half saving, so to speak, the affected half, there is a virtual paralysis of the internal rectus of the non-paralysed side. But this is for a time only; the volitional influence soon passes round the other way, and both eyes recover their full freedom of movement. Something of the same kind may often be witnessed in the orbicularis oculi. At first this is weakened, the communication between the two nuclei is not complete; but this is speedily perfected, and the loss of power ceases to be apparent. That the influence here again comes round by the nucleus of the non-paralysed side is demonstrated by bidding the patient to wink the eye of the hemiplegic side alone. He will be unable to do this and will probably close the other by itself in the attempt.

To go back to the deviation of the eyes. It may be understood how this occurs if we reflect that, in turning the eyes to either side, the eye of that side seems to lead, the other following (best exemplified when the outer eye is fixed on an object which the other cannot see on account of the nose). In other words, the external rectus habitually receives the volitional impulse, the internal rectus of the other eye acting consensually with it, and not under the direct influence of volition. When, then, the external rectus is abruptly cut off from the volitional centre of its own side, if it acts at all, it

must be consensually subordinate to the opposite internal rectus now directly under the influence of the will. This reversal in the course of the nervous current is not, in all cases, at once brought about, and until it is the deviation exists.

[It is interesting that, though it is noted that the head was turned to the left, no mention is made of conjugate deviation of the head and eyes.—Ed.]

EXTRACT FROM A LECTURE ON CONJUGATE DEVIATION OF THE HEAD AND EYES, AS A SYMPTOM IN CEREBRAL HAEMORRHAGE AND OTHER AFFECTIONS

The Lancet, 1879

I HAVE told you that this deviation of the head and eyes is almost always a temporary thing; but if you get a certain lesion in the pons the deviation is permanent. In a case reported by me to the Clinical Society (*Trans.*, vol. v.) there were various evidences of lesion in the pons, one of which was persistent deviation of the eyes to one side. The patient was an old soldier who had suffered from syphilis. He walked feebly and staggered, but moved both legs equally well. There was no obvious loss of power in either arm or hand; and though he said the right hand felt dead, sensation, when tested, did not appear to be much affected. There was thus no hemiplegia of the limbs, and the tongue was protruded straight, but the left side of the face exhibited motor paralysis, while over the right half sensation was markedly impaired; the soft palate, moreover, was paralysed, causing the articulation to be nasal and indistinct, while deglutition was difficult. The paralysis was not the "facial paralysis of Bell" but "facial hemiplegia," not affecting the orbicularis oculi, and it was associated with lateral deviation of the eyes to the right, which was conspicuous and permanent throughout his illness of two months. Had there been paralysis of the portio dura and of the sixth (complete paralysis of the left face with unclosing eye, and internal squint) the case would have been simply one of lesion affecting the combined nuclei of the portio dura and sixth; but the condition was altogether different. The diagnosis made was syphilitic tumour in the pons and medulla; and after death, which was sudden, two small tumours were found in the middle line near the floor of the fourth ventricle: one in the medulla, which would cause the difficulty of deglutition; the other in the pons, exactly in the situation to cut off the communication between one third nerve-nucleus and the other sixth, while the facial hemiplegia and the ocular deviation showed that the fibres from the hemisphere to the facial and sixth nuclei were divided. It had rendered impossible the reversing of the current, so the deviation became permanent.

ON THE STRUCTURE OF THE CEREBRAL HEMISPHERES

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(*Abstract*)

THE object of the investigation has been twofold. First and chiefly, to endeavour to ascertain minutely the course of the fibres by which the convolutions of the hemisphere are connected with each other and with the crus and central ganglia.

Secondly, to ascertain whether there is a constant similarity between the corresponding sides of different brains as compared with the opposite sides of the same brain; and should this be the case, to endeavour to trace the relation between any anatomical difference which might be discovered and such physiological difference as seems in the present state of our knowledge to be indicated by the association of loss of the faculty of language with disease of the left hemisphere rather than the right.

The present communication relates almost exclusively to the first branch of the investigation, and the method pursued has been to harden the brain by prolonged immersion in strong spirit, by which the fibres are rendered perfectly distinct and fairly tenacious, so that with care and patience their course and arrangement may be accurately ascertained.

Previous researches on the structure of the cerebrum have been mainly directed to the examination of the course and distribution of the fibres radiating from the crus and central ganglia, which have been assumed or supposed to occupy ultimately the axis of every convolution, the different convolutions being connected by fibres which crossed under the sulci from one to another. It is here shown that the commissural communication between different parts of the hemisphere is much more extensive than has hitherto been described, and that the fibres more commonly run longitudinally in the convolutions than cross from one to another, while large tracts of convolutions have no direct connexion with the crus, central ganglia, or corpus callosum.

The preponderance of commissural over radiating fibres is indicated by a comparison of the sectional area of the latter as they issue from the central ganglia with the large surface of white matter displayed in the centrum ovale. The dissection by which

this is shown in detail is begun on the under surface of the temporo- or occipito-sphenoidal lobe.

Dissection of the Temporo-Sphenoidal Lobe.—In this lobe the fibres are almost entirely longitudinal in their general direction. From near the apex fibres can be followed backwards in the two or three convolutions on the outer side of the gyrus uncinatus to near the centre of this surface of the lobe, where they end in the grey matter of a sort of lobule which I have ventured to call the *collateral lobule.*[1] From the collateral lobule other fibres pass to the convolutions at the occipital extremity of the lobe, to convolutions on its outer side and to the calcarine end of the uncinate gyrus. These convolutions, comprising all those of the temporo-sphenoidal lobe except the gyrus uncinatus, the inframarginal and parallel gyri, and the continuation of the two latter round the apex, receive no fibres whatever from the crus, central ganglia or corpus callosum, but the anterior commissure spreads into them.

Beneath these is a beautiful plane of fibres which forms the floor of the descending cornu of the lateral ventricle, except at the anterior end; it forms the floor also of the ventricle at the entrance to the cornu, i.e. in the eminentia accessoria, and of the posterior cornu; but here fibres of the corpus callosum are mingled with those of the plane spoken of. This plane is formed as follows: along the axis of the lobe, in the hollow left by the removal of the superficial convolutions runs a band of fibres from the apex to the posterior extremity; anteriorly this band contains numerous fibres, but in passing backwards they spread out towards the inner border of the lobe into a continuous lamina, which rests upon the lining membrane of the ventricle and its cornua. Some of the fibres run in the upper wall of the calcarine fissure to the postero-parietal lobule, others form a layer in the lower wall of this fissure, i.e. in the calcarine division of the gyrus uncinatus.

The Gyrus Uncinatus remains as an elevation along the inner side of the shallow valley resulting from the dissection described, little encroached upon by it; its superficial fibres, however, must be removed to display the plane just mentioned. It encloses the cornu of the ventricle and the hippocampus, and is thus not a solid mass. Its fibres can be divided into two layers, a superficial set, the general direction of which is from the outer or collateral side anteriorly, backwards and inwards to the grey matter on its flat surface; and a deeper set, the fibres of which at the anterior part of the gyrus occupy its entire width, in passing backwards they converge, and near the inner border have a twisted arrangement, the inner fibres passing

[1] *Vide* p. 290.

beneath the outer to the grey matter of the hippocampus and to the splenium of the corpus callosum, the outer fibres crossing over and reaching the upper wall of the calcarine fissure, in which they pass to the postero-parietal lobule and to the callosal gyrus.

The anterior enlarged extremity of the uncinate gyrus, sometimes called the uncinate lobule, is connected by bands of fibres with various parts ; it is very firmly adherent to the subjacent structures and when torn away leaves a patch of exposed grey matter, which has been named the internal grey nucleus. This is about in the same transverse line with the corpus albicans, a little to the outer side of the optic tract.

By the removal of the uncinate lobule and gyrus, fibres can be seen to pass from the apex of the lobe forwards in the fasciculus uncinatus, backwards and inwards along the roof of the cornu to the thalamus, and inwards to the grey nucleus.

On further Dissection, which will consist in tracing the fibres from the apex backwards to various parts, and in removing little by little more of the convolutions along the outer edge of the lobe, and in a careful investigation of the parts about the calcarine fissure, the following appearances will be presented :—

Along the axis of the lobe a longitudinal ridge with a slight convexity outwards, prominent posteriorly, subsiding anteriorly. On its inner side, from behind forwards, first the posterior cornu ; next the outer wall of the ventricle, where the cornua enter it ; this is formed by fibres curving directly backwards into the ridge from the thalamus (also from crus and corpus striatum, but more deeply), they are crossed transversely, however, by a thin lamina of fibres from the under surface of the splenium, which bend down from the roof of the ventricle and then curve forwards in the ridge ; next the posterior end of the thalamus, which bends forwards round the crus, and gives off forwards from a pointed extremity the optic tract and laminae of fibres on the outer side of this, which run above the roof of the cornu to the apex. Anteriorly this longitudinal ridge is continuous with the fasciculus uncinatus, and on its inner side are the internal grey nucleus, and more anteriorly the anterior perforated space between which the anterior commissure dips forwards and inwards in its canal.

On the outer side of the ridge fibres may be seen to start at the edge of the lobe, run inwards to the ridge, and curve forwards in it, to leave it again on its outer or inner side, or to pass with it to the fasciculus uncinatus.

A bundle of fibres taken up from the posterior part of the ridge would pass mainly to the thalamus ; but some would proceed forwards in the ridge, and either turn outwards to some part of

the inframarginal gyrus or apex, or inwards to the internal grey nucleus, or behind it. Others again go on in the fasciculus uncinatus.

Fibres taken from the middle part of the ridge, and traced backwards would mostly curve outwards to some part of the outer edge of the lobe, but some would go to the tip; followed forwards, they spread out into a thin fan, and pass to the various points already indicated.

By repetition of this process the temporo-sphenoidal lobe will be exhausted, with the exception of a considerable lamina of fibres from the posterior part of the inframarginal gyrus, which passes backwards and inwards to the end of the fissure of Sylvius, round which it curves into the supramarginal gyrus, and another large band from the posterior end of the parallel gyrus, which curves upwards and turns forwards in the axis of the parietal lobe close behind the fibres which curve upwards from the corpus callosum to the margin of the longitudinal fissure.

It should be added that large bands of fibres run obliquely backwards in the parallel gyrus to the bottom of the sulcus of the same name, under which they turn to the inframarginal gyrus. When these are removed the deep parallel sulcus is converted into a deep narrow valley.

The Fasciculus Uncinatus, in the dissection just described, has been seen to receive fibres from the occipital extremity of the hemisphere, and from the various convolutions along its outer side, occipital, annectent, angular, parallel, and inframarginal; fibres are traceable into it also from the internal grey nucleus, these mostly lying beneath those from the convolutions, and it is probable that a few fibres from the thalamus and splenium find their way into it. As it emerges from under the temporo-sphenoidal lobe to cross the entrance to the fissure of Sylvius, it receives a considerable contribution from the overhanging apex of this lobe, and some from the uncinate lobule. Its general direction is forwards; but a superficial set of fibres mainly from the apex of the temporo-sphenoidal lobe, passes inwards as well as forwards, and spreads out mainly to the edge of the longitudinal fissure, passing under the olfactory sulcus; another lamina appears from beneath the edge of this, having a still more transverse direction, and its fibres go to the rostrum corporis callosi, and to the callosal gyrus, detaching the pointed origin of this convolution from the anterior perforated space. The fibres passing directly forwards spread out under the orbital convolutions to end in the grey matter around the edge of this lobule, some of the more superficial turning into one or two of the gyri at its posterior and outer margin. Deeper fibres run outwards as well as forwards, beneath the convolu-

tions of the island of Reil, to the posterior part of the inferior frontal gyrus; this is a tract of considerable size.

The convolutions of the *orbitar lobule* being entirely superficial to the radiating fibres of the fasciculus uncinatus, must be added to those on the under surface of the temporo-sphenoidal lobe as belonging to the class which have no direct central communications.

The Gyri Operti.—To this class also must be added, with a reservation to be noted presently, the gyri operti of the island. The summit and the anterior convolutions rest upon the part of the fasciculus uncinatus which passes to the outer corner of the orbitar lobule and the third frontal gyrus, and the fibres arising in the grey matter of this portion of the island curve forwards across the fissure to the same convolutions; the corner of the orbitar lobule in fact is carried away entirely by the fibres from the fasciculus and island. In the same way fibres starting in the remaining convolutions of the island cross the fissure and turn up in the supramarginal gyrus, leaving the outer surface of the corpus striatum perfectly smooth, and converting the Sylvian fissure into a deep wide valley. The wall of the corpus striatum thus exposed consists of a lamina of fibres, which radiate in all directions from a small patch of grey matter laid bare at the middle and highest point of the eminence this ganglion forms as seen from this aspect; and it is possible that there may be here some sort of continuity or connexion between the grey matter of the corpus striatum and the overlying part of the convolutions of the island. Except at this point, the convolutions are separated from the corpus striatum by a very distinct plane of fibres.

The gyri operti are thus connected mainly with the supramarginal gyrus and its continuation along the anterior wall of the fissure. Some fibres, however, pass from the grey matter of the overhanging inframarginal gyrus near the apex into the corresponding part of the island, and, about the grey nucleus exposed at the summit of the corpus striatum, deep fibres from the posterior extremity of the hemisphere and from the fasciculus uncinatus seem to join both the nuclues and the overlying grey matter of the island.

The temporo-sphenoidal lobe having been gradually removed, and with it a great part of the occipital lobe, a stage of the dissection is reached at which the distribution of the fibres of the splenium corporis callosi and the relations of the crus and central ganglia, as seen from the under aspect, may be conveniently described.

The Splenium Corporis Callosi.—On the inferior surface of the posterior extremity of the corpus callosum is seen a transverse flattened elevation, which may be compared to the rostrum at

the anterior extremity on a smaller scale and adherent to the body of the great commissure. It would thus be looked upon as a recurved part of the corpus callosum. In the middle line it is adherent, but the fibres it sends transversely outwards leave the corpus callosum proper, and bend downwards so as to cross the floor of the ventricle instead of the roof; they pass to the hippocampus major and minor, which they contribute to form, and run across the eminentia accessoria, and along the floor of the posterior cornu.

The Hippocampus Minor is formed by the projection into the posterior cornu of the bottom of the calcarine fissure; but an incision through the bottom of the fissure into the cornu would not split up the hippocampus, but would leave it attached entire to the upper wall of the cornu. The fibres from the splenium, which contribute to the formation of the hippocampus minor, run longitudinally along it immediately beneath the lining membrane of the ventricle, and, when reached by dissection from without, present a delicate lamina in the form of a groove between two curved tracts passing backwards to the posterior extremity of the hemisphere, the upper from the corpus callosum proper, the lower from its recurved process.

The Hippocampus Major may be briefly described as a curved groove or "gutter" (Gratiolet) of fibres, the upper border of which is formed by the posterior pillar of the fornix, while the lower is concealed by the gyrus uncinatus, the grey matter of which folds over it into the groove, and after reaching the bottom bends up the other wall for a short distance, forming the plicated "corpus fimbriatum" or "pli godronné." The outer surface of the case of fibres is smooth, and for the most part free in the descending cornu; it adheres to the inferior wall formed by the plane of fibres previously described, but can easily be detached. The course of the fibres forming the case or groove is from the lower edge backwards and upwards round the convexity to the upper edge, where they pass into the pillar of the fornix, or where the hippocampus joins the splenium, into the recurved process. Further details are given in the paper itself.

The fibres crossing the floor of the ventricle curve forward, apparently towards the apex, but are too few to be followed absolutely to their termination.

From the body of the corpus callosum, at its posterior part, the fibres mostly radiate backwards and outwards into the cuneus and occipital lobe generally; but a considerable number on the under surface bend from the roof of the ventricle down its outer wall across the longitudinal fibres from the thalamus, etc., and

curve forwards in the ridge. A considerable proportion of these has been traced to the internal grey nucleus; others seem to pass forwards to the grey matter near the apex of the temporo-sphenoidal lobe.

The Crus and Great Central Ganglia.—The relations of the crus and great central ganglia may be described as follows:—The crus, as it plunges into the hemisphere, is encircled on its inferior aspect by the optic tract, it then expands into a large fan of fibres, the edges of which are antero-posterior, the surfaces obliquely upwards and inwards, and downwards and outwards. The two great ganglia, the corpus striatum and thalamus, may be said to sit astride the anterior and posterior edge respectively of the fan, each having an intra and extra-ventricular part, the corpus striatum being much the larger, and situate above, as well as in front of the thalamus.

When the optic tract is removed, the groove in which it rests is seen to present fibres having the same general direction round the crus; they have been called by Gratiolet "l'anse du pédoncle," a term which may be translated by the expression "the collar of the crus." The most conspicuous part of the collar consists of fibres from the thalamus, which curve forward round the crus to end in the tuber cinereum, or run up in the wall of the third ventricle to the velum interpositum, etc. Within this fibres are seen to turn forwards from the posterior border of both crust and tegment of the crus, to end in the corpus striatum, and anteriorly a considerable mass of fibres from the tegmentum curves with a bold sweep round the edge of the crust, and passes backwards and outwards into this same ganglion.

The extra-ventricular part of the thalamus is seen in the descending cornu curving round the crus. From its anterior pointed extremity it is continued onwards by the optic tract, and it sends fibres—(1) Forwards in the collar of the crus. (2) Forwards and outwards to the convolutions about the apex in a succession of laminae, the deeper fibres passing more outwards than the superficial sets, and emerging from under them along the outer edge of the roof of the cornu. (3) From under the fibres which pass forwards, it sends backwards a large mass along the outer wall of the ventricle and posterior cornu to the occipital end of the hemisphere.

The extra-ventricular corpus striatum has been exposed on two sides; it forms a very large mass, and has a large rounded anterior end, while posteriorly it narrows to a tail-like extremity. The outer aspect forms an elongated eminence, rising out of the Sylvian valley highest at the centre, subsiding towards each end; at the summit is the external grey nucleus, from which radiate fibres

forwards, backwards, and outwards. Those passing forwards form a large bundle; they spread out into a fan, and proceed mainly to the third frontal convolution; those passing backwards accompany the fibres from the thalamus to the occipital extremity of the hemisphere; those passing outwards with varying degrees of obliquity descend the wall of the ganglion to the Sylvian valley; but instead of crossing it to the convolutions on the other side, as might be expected from the apparent continuity of the walls and floor, dip between the fibres of the floor, which are the radiating fibres of the crus issuing from the corpus striatum, and passing to convolutions in the frontal lobe.

A remarkable fact respecting the planes of radiating fibres which form the limiting wall of the corpus striatum on this aspect is, that the fibres all seem to have their origin in the small patch of grey matter here called the external grey nucleus, and they come off clean from the mass of soft grey matter forming the body of the ganglion.

On the under surface of the corpus striatum, which is flat, are seen the internal grey nucleus and the anterior perforated space, between which the anterior commissure passes outwards and backwards from the ventricle in a distinct canal to emerge on this surface. The external grey nucleus also appears in the outer border, and is about in the same transverse line as the corpus albicans and internal grey nucleus, from which last it is only separated by a narrow band of longitudinal fibres. Here again the planes of fibres, which form the limiting wall of the ganglion, end in the grey nuclei, and seem to have no communication with the mass of soft grey matter they inclose.

The anterior edge of the fan-like expansion of the crus emerges from the large end of the corpus striatum, and, properly speaking, divides the intra-ventricular corpus striatum from the extra-ventricular division; the anterior perforated space, being on the inner side of the radiating fibres, belongs to the former.

Before the dissection of the fronto-parietal portion of the hemisphere is described, a brief account is given of the intra-ventricular thalamus and corpus striatum.

When the taenia semicircularis is removed, and the edge of the corpus striatum pushed back, large rounded cords of fibres are seen radiating outwards in all directions from the thalamus with the fibres of the crus, posteriorly slender flat bands of fibres curve backwards from the narrowing extremity of the corpus striatum to dip down between them (together with fibres apparently belonging to the taenia); they can be traced through the fan of radiating fibres to the extra-ventricular corpus striatum. Anteriorly the

soft grey matter of the corpus striatum fills the spaces between the diverging cords; but no distinct origin of fibres in the mass of grey matter is here met with.

The Frontal and Parietal Lobes.—The plan of construction of the fronto-parietal portion of the hemisphere seems to be as follows:—

The corpus callosum divides into two main planes of fibres, one of which turns up to the margin of the great longitudinal fissure, the other passes onwards to the supramarginal gyrus of the fissure of Sylvius. The radiating central fibres approach the under surface of these at an acute angle, and pass obliquely between them before the ascending and descending planes have well separated from each other, the central as well as the callosal fibres going mainly to the margins of the hemisphere. An angle is thus left along the axis of the frontal and parietal lobes, which is occupied by a vast longitudinal system of fibres, some of which have already been mentioned as entering this part of the hemisphere from the temporo-sphenoidal lobe. Large bands turn upwards and then forwards from the parallel and angular gyri, that from the parallel gyrus running forwards close behind the ascending callosal lamina; other fibres turn forwards from the annectent gyri, and more anteriorly from the postero-parietal lobule; still further forwards some of these fibres coming from behind bend upwards, and end in the parietal convolutions; while others start in the same gyri, and pass forwards, the principle of construction being apparently simple, but the details extremely intricate.

At the decussation the central and callosal fibres are woven into a compact inextricable mass, and the difficulty of following the different sets is increased by the fact that the central fibres are not transverse in direction like those of the corpus callosum, but mostly very oblique backwards or forwards, as may best be seen by examining the bands radiating under the corpus striatum from the thalamus; this necessitates corresponding obliquity in the fissures through which the central fibres penetrate the corpus callosum. A few fibres from the under surface of the corpus callosum turn inwards to the centres; but the statement of Gratiolet that all the fibres of this commissure can be traced from the central radiations on one side to the convolutions on the other, is not confirmed.

The detailed dissection of the parieto-frontal convolutions need not be given here. It will be sufficient to mention that the postero-parietal and supramarginal lobules are connected by numerous bands of fibres, that the ascending parietal gyri have central and callosal fibres entering their extremities, the middle portion receiving comparatively few; the first, however, sometimes called the ascend-

ing frontal gyrus, seems to have numerous fibres from the centres and corpus callosum along its entire length. The second frontal convolution sends bands of fibres obliquely to the two others, and has fewer radiating fibres than they have. When it is removed, fibres can be traced transversely across the valley left from the first to the third.

A few additional particulars are given respecting the arrangement and course of the fibres in the callosal and marginal gyri on the internal surface of the hemisphere, and the contrast between the thalamus and corpus striatum as to structure and relations is pointed out, the thalamus sending large masses of fibres in every direction, chiefly with the radiating crus, the corpus striatum consisting of soft grey matter enclosed in fibrous planes which arise in the comparatively small grey nuclei, and have apparently no communication with the main body of the ganglion. The thalamus again does not seem to receive terminating ascending fibres, while both divisions of the crus give off numerous fibres, which are seen to end in the corpus striatum.

The differences in naked-eye appearances indicate differences in the relations between cells and fibres in the two ganglia, the exact nature of which can be ascertained only by the microscope.

ON THE CEREBRAL CONVOLUTIONS OF A DEAF AND DUMB WOMAN

Journal of Anatomy and Physiology, 1870

THE following are some of the objects sought to be attained by a careful study of the convolutions of the cerebral hemispheres.

1. To ascertain if there is any constant difference between the right and left sides of the brain, which may throw any light on the apparent difference in function exemplified in the association of aphasia, so called, with injury of the left frontal lobe.

2. To trace the gradual development of the characters which distinguish brains capable of the highest intellectual operations.

From this point of view it will be interesting to examine the cerebrum in individuals belonging to the Negro, Mongol, and other pure races, so as by comparison of one with another, and of all with the brains of the Bushwomen figured by Gratiolet and Marshall, and with the brains of the primates generally, to ascertain how far the development has been linear and general or divergent and partial.

It will be interesting also to examine the cerebral hemispheres of new-born infants and of young children at different ages, so as to learn how far the subsequent growth is by simple enlargement of convolutions present at birth, how far by the interposition of new convolutions. A comparison of infantile brains of the intellectual classes and of the classes subsisting by manual labour would be of course necessary to the elucidation of this question.

3. To endeavour to connect varying degrees of intellectual power and peculiarities of mental constitution with peculiarities or differences in the arrangement and complexity of the convolutions. This would be to construct a scientific phrenology. The brains of idiots will be of great use in this part of the investigation. It has always seemed to me a mistake to select for examination and representation the extremely degenerate brains of the lowest idiots; the most valuable results will be obtained from those cases just on the one or other side of the line of idiocy, or in which some mental faculty is abnormally predominant.

4. Still another object which led me to seek for the brains of

deaf and dumb or congenitally blind individuals will be explained by a quotation from a paper by Dr. Bastian, "On the Muscular Sense, and on the Physiology of Thinking." "I start," he says, "with the presumption that in the higher phenomena of mind, which are dependent upon the cerebral hemispheres, certain definite parts of these are always called into activity whenever similar mental operations are repeated. . . . I also assume that the several sense-centres at the base of the brain and in the medulla are connected in a perfectly definite way, each with its own set of cells in the cortical substance of the hemispheres; these cells, in connexion with the several sense-centres constituting their respective *perceptive centres*, which may exist in regions of the hemispheres either distinct from one another, or which may be variously interblended. In the perceptive centres, the primary impressions made upon the organs of sense are converted into 'perceptions' proper; that is to say, they receive their intellectual elaboration, and this elaboration implies an intimate cell and fibre communication between each perceptive centre, and every other perceptive centre, since one of the principal features of a perceptive act is that it tends to associate as it were into one state of consciousness much of the knowledge which had been derived at different times and in different ways concerning any particular object of perception. An impression of an object, therefore, made upon any single sense-centre, on reaching the cerebral hemispheres, though it strikes first upon the corresponding perceptive centre, immediately radiates to other perceptive centres, there to strike upon functionally related cells, all this taking place with such rapidity that the several excitations are practically simultaneous, so that the combined effects are fused into one single perceptive act."

Speculations similar to these had, previously to the appearance of Dr. Bastian's paper, suggested to me the idea that in the brains of the deaf and dumb and of the congenitally blind, there might be unused areas or tracts of convolutions which might in consequence have failed to undergo development corresponding to the general development and growth of the hemisphere. In this case the cerebral convolutions of the deaf and dumb and blind might be expected to exhibit more or less constant and definite peculiarities, which by examination and comparison of many examples might be made out. The deaf and dumb, again, are deprived of the faculty of giving expression to their ideas by articulate language; a most important "way out" for the products of intellectual operations, to adopt a term introduced by Dr. Hughlings Jackson, therefore remains unused, and the convolutions which serve as the downward starting-point of the ideo-motor impulses which

give rise to spoken words might be expected to stop short in their growth. At any rate since the blind and deaf each specially cultivate perceptive faculties of which the others are deprived, and since the entire basis of their mental operations must be different, a comparison of their cerebral hemispheres with each other and with those of persons possessing both sight and hearing cannot fail to be interesting.

In the brains I may have the opportunity of examining personally, I shall endeavour to connect any modification in the arrangement of the convolutions exhibited on the surface, with the concomitant modification in the course and connexions of the fibres in the substance passing between the crus and central ganglia and the cortical grey matter on the one hand, or between different convolutions on the other. I may state that repeated and careful dissections have led me to the conclusion that the fibres radiating from the crus and central ganglia are distributed mainly to the two extremities of the hemisphere and along the upper and outer edges, i.e. along the margin of the great longitudinal fissure above, and along the third frontal gyrus and the upper edge of the fissure of Sylvius below, the fibres of the corpus callosum passing mainly to the same parts though in different proportion. Of the remaining convolutions it may be said with certainty that some receive no fibres whatever from the crus, central ganglia, or corpus callosum, e.g. those on the internal flat surface of the hemisphere, those of the orbital lobule except at the margin, those of the under surface of the temporo-sphenoidal lobe, except around the edge, and those of the Island of Reil. Those on the convex outer surface of the hemisphere receive few as compared with the two margins. The mass of white substance is composed of fibres passing from one part of the hemispherical surface grey matter to another in large commissural systems, and the convolutions indicate, not as Gratiolet states, the distribution of the central radiating fibres, but the course of the superficial commissural or "proper" fibres.

It at once occurs to the mind that the convolutions in direct relation with the crus and central ganglia will constitute the "perceptive centres" on which impressions travelling upwards first impinge, and the downward starting-point of volitional impulses, while those convolutions which are withdrawn, so to speak, from immediate relation with the outer world, will be the seat of the more purely intellectual operations; and it is in effect in the superadding of these gyri that the difference between the higher and lower primates, and between man and the primates consists.[1]

[1] For fuller details see the *Proceedings of the Royal Society* for July, 1869 and *Journal of Mental Science* for April, 1870.

The first brain to be described is that of a deaf and dumb woman who died from accident in the Middlesex Hospital under the care of Mr. C. H. Moore, to whom I am indebted for the opportunity of examining it.

The brain was of the full average size for a woman, weighing on its removal from the cranium 45 oz. It presented nothing remarkable in its general appearance and conformation. It was flattened by its own weight before it came into my possession: the convolutions therefore appear in some degree distorted in the tracings.[1]

External Aspect of Fronto-parietal and Occipital Lobes.
Fissures and Sulci.

1. Fissure of Sylvius, *SS*. Right. Ascending branch short; interrupted by origin of third frontal gyrus from anterior parietal; horizontal branch comparatively straight, its extremity surrounded by a remarkable "angular" convolution.

Left. Ascending branch longer; reaches second frontal gyrus, having in it a sort of island which is a portion of the third frontal partially cut off. Transverse branch extends rather farther back than on the right side, a convolution curves round its termination, but superiorly expands into a supramarginal lobule.

2. Sulcus of Rolando, *RR*. Right extends at about the usual slope from near the fissure of Sylvius to near the median fissure. Left has its lower end nearer the Sylvian fissure, but its upper not so close to the edge of the hemisphere.

3. Intra-parietal sulcus, *IP*. Right does not reach the fissure of Sylvius below, is intercepted by the angular convolution mentioned; above extends to near the edge of the hemisphere. Left

[1] The tracings were made by the following method, practised with great success by Dr. Sibson, from whom I learnt it, in figuring the fibres of the heart. A sheet of glass is placed over the brain as close to it as possible; the fissures and sulci are traced upon the glass in Indian ink or some other pigment by means of a camel-hair pencil, the eye being carefully maintained perpendicularly over the point to be represented. If this precaution is not taken, and especially if the glass is not equidistant from every part of the object or is at some little distance above it, the result may be very erroneous. The tracing is then transcribed on thin paper placed over the glass and held up to the light, and is afterwards carefully compared with the brain so as to eliminate accidental and unimportant markings and to give due relative importance to the different fissures and sulci. Of course the convolutions appear wider than they would in a shaded drawing, but a degree of relative accuracy is obtained which will permit of comparisons being made between different brains and between different parts of the same brain. Both hemispheres are figured and described, the association of aphasia so called with disease of the left frontal lobe having given a new motive for comparing the two halves of the brain.

bends forwards at its lower end into the second ascending parietal gyrus, does not reach the median fissure.

4. External parieto-occipital fissure, *PO*. Right wide and extending nearly to the parallel sulcus, cutting off the occipital from the parietal lobe. Small annectent gyri are however concealed within it.

Left very short, being at once intercepted by the first annectent convolution, but close to its termination begins a deep sulcus, *PO*, which runs down to the parallel sulcus and separates the parietal and occipital lobes. This sulcus is crossed midway by another, the two forming a cruciform marking, which at once arrested the attention.

Convolutions.

1. Frontal *1, 1*; *2, 2*; *3, 3*. Right. First and second large, and each has two origins from the first parietal; the first is single but wide, the second is double. Third springs from first parietal, and makes two bends upon itself before turning forward along the lower edge of the lobe.

Left. First and second have each a single origin from the anterior ascending parietal with a small intercalated portion of gyrus between them. The first is single throughout and rather small; the second becomes double in passing forwards and is rather large. The third does not spring from the ascending parietal. A part is nearly cut off from its posterior extremity, and forms a small island seen in the ascending branch of the fissure of Sylvius. Including this it is smaller than the right third gyrus.

2. Parietal. The ascending parietal convolutions, one *4*, in front of, and the other *5*, behind the sulcus of Rolando, present no features calling for remark. Round the end of the fissure of Sylvius on both sides is a curved convolution, and extending from the lower end of the second parietal to the inframarginal convolution of the fissure in the temporo-sphenoidal lobe; this takes the place of the supramarginal lobule, and apparently also of the angular gyrus or "pli courbe." The postero-parietal lobule, *pp*, has no obvious peculiarity.

3. Occipital. The convolutions of the occipital lobe seemed to be simpler than usual; the annectent or bridging gyri deficient.

Temporo-sphenoidal Lobe.

Fissures and Sulci.

1. Calcarine fissure, *Cc*. Right anteriorly extends nearly across the gyrus uncinatus; posteriorly is curved and passes out on extremity of occipital lobe, and appears on the under surface at

the tip. A bifurcation runs upwards. Left, does not cut across the uncinate gyrus; has a straight course, and at the top of the occipital lobe curves to the inferior aspect.

2. Collateral sulcus, *Cl.* Right interrupted anteriorly, bifurcates posteriorly, the outer branch apparently being the continuation of the sulcus, and falling into a curved transverse sulcus near the extremity of the lobe. Left not interrupted anteriorly; its bifurcation encloses a larger gyrus, and its inner branch continues backwards nearly parallel with the calcarine fissure.

3. A lateral sulcus, *L* (not named) is deep and uninterrupted on the right side, much broken up on the left.

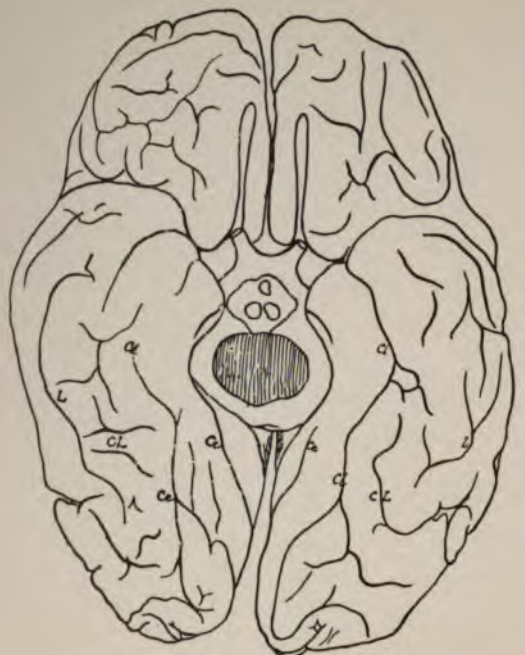
4. Parallel sulcus, *P*. Right uninterrupted; has a simple termination posteriorly, but the sulcus surrounding the curved gyrus round the end of the fissure of Sylvius falls into it. Left interrupted anteriorly; posteriorly runs into the large cruciform sulcus mentioned.

Gyri. The only point calling for remark is the small size of the collateral lobule *CL* on the left side. (This lobule is situate nearly over the situation of the eminentia collateralis between the collateral and lateral sulci, and has been named because in it fibres end which come from the two extremities of the lobe and from the convolutions on each side. The sulci bounding it dip under it so that with a wide surface it has a slender deep attachment.)

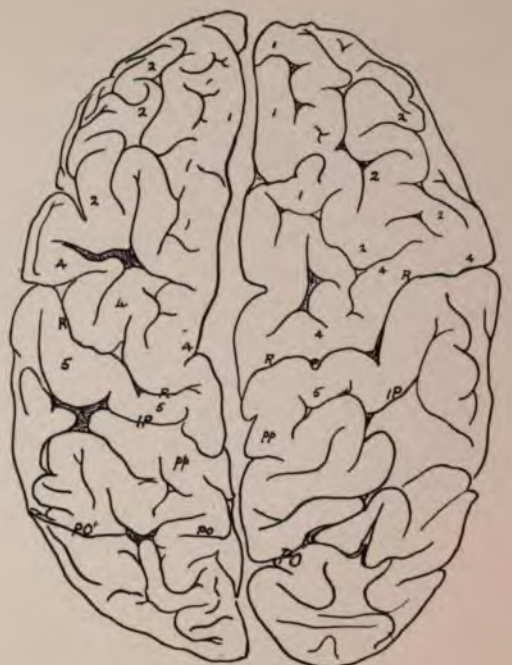
Island of Reil. Convolutions arranged in three pairs as usual, apparently somewhat smaller and more simple than usual. No difference noted between the two sides.

The convolutions of the orbital lobule present no peculiarity, they are rather more simple on the left side which, according to my observation, seems to be the rule.

The Callosal (*Cal.*) and Marginal (*Mar.*) Gyri on the internal flat surface, the Quadrilateral Lobule (*Q*) and Cuneus, (*Cu*) do not require description, but attention may be called to an arrangement of the commencing callosal and marginal gyri of the two sides which is met with in all brains. At this part, i.e. in the median fissure below the rostrum of the corpus callosum, the falx is not interposed between the two hemispheres, and they not only lie in contact with each other but are adherent, and the convolutions of one fit into the sulci of the other. In the tracing it will be seen that the marginal gyrus of the left hemisphere is subdivided into three narrow folds by two sulci (the upper of which is deep, the lower shallow). The central fold was very prominent, and occupied the groove formed by the sulcus which runs along the corresponding gyrus of the right side; above and below it were grooves, into which fitted prominences on the left hemisphere; and so as to the callosal gyrus.



Under Surface.



Upper Surface.

PLATE I.

BRAIN OF DEAF AND DUMB WOMAN.

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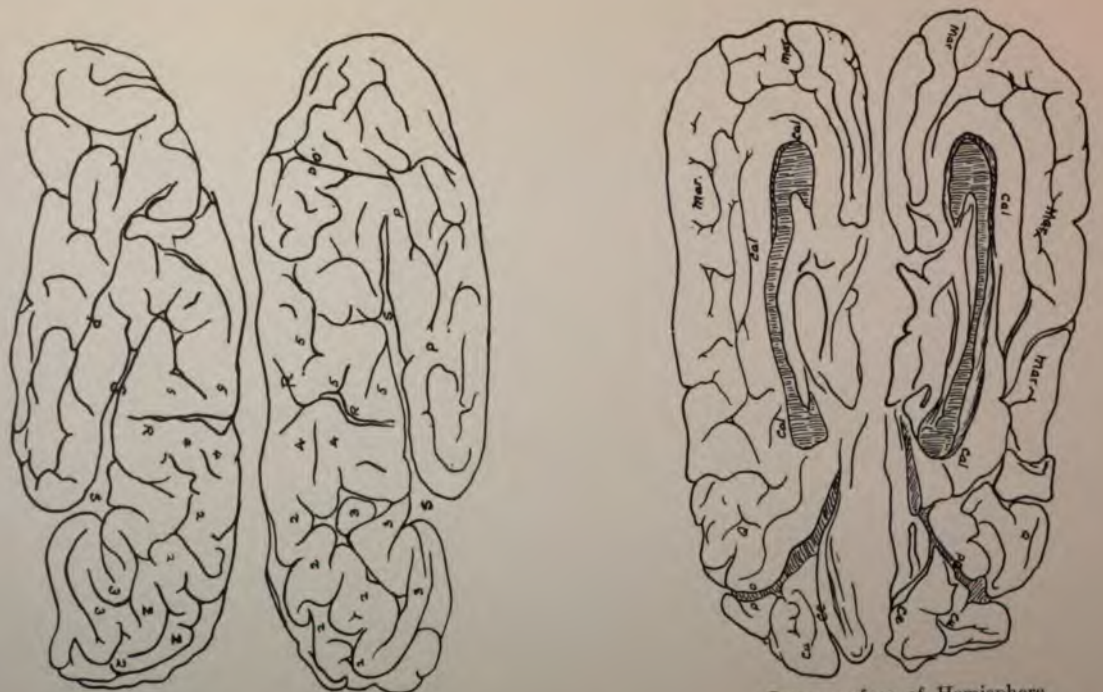

Inner surface of Hemisphere.

Right Hemisphere.
(Outer aspect.)

Left Hemisphere.
(Outer aspect.)

PLATE II.

BRAIN OF DEAF AND DUMB WOMAN.

[To face page 291.

Summing up the points in which peculiarities have been noted in this brain, they are the comparatively small size and simple character of the left third frontal gyrus ; a deficiency of annectent convolutions, so that the occipital lobe is almost completely separated from the parietal ; a deficiency in the supramarginal lobule, which allows the angular gyrus usually displaced backwards to surround the end of the fissure of Sylvius ; a degree of simplicity of the occipital lobe generally and a narrow collateral lobule in the temporo-sphenoidal lobe. (In the tracings of the brain the left superior temporo-sphenoidal convolution is smaller than the right.) These may all be merely features of general inferiority common to many brains, or perhaps some of them may be specially associated with deaf-mutism. This can be ascertained only by the examination of numerous cases. The small size of the third frontal gyrus on the left side of course challenges attention, but while signalizing the fact, I attach no importance whatever to it unless it is corroborated by other observations. The absence of annectent gyri and the almost complete severance of the occipital lobe from the parietal by the external parieto-occipital fissure on the one side, and by the sulcus prolonged upwards from the parallel sulcus on the other, are certainly remarkable but not altogether peculiar. I am inclined to look upon this condition as in some degree a feminine characteristic. The comparative symmetry and simplicity of the convolution round the end of the fissure of Sylvius constitute another feature worthy of note. The small size of the collateral lobule would probably have escaped attention had it not been attended with a remarkable simplicity in the arrangement of fibres. The one point which seems to have a certain importance is, that the degree of convolutional development does not seem to be equal at the two ends of the hemisphere ; while the frontal gyri are elaborate and bridge over the ascending branch of the Sylvian fissure, so as to connect the frontal and parietal lobes ; the convolutions about the parieto-occipital fissure and the posterior end of the Sylvian fissure are comparatively simple and permit of an unusual breach of continuity between the parietal and occipital lobes.

A LECTURE ON THE THEORY OF CONSTRUCTION OF THE NERVOUS SYSTEM

Delivered at the West Riding Asylum, Wakefield

British Medical Journal, 1876

THE word theory is not altogether free from objection, as here employed; but, as the facts of structure have to be interpreted by experiment, the conclusions reached, however definite and certain they may ultimately become, can scarcely be other than a theory.

The Spinal Cord.—We must begin as usual with the spinal cord, which is not only the instrument of the brain and the medium of communication between it and the outer world, but offers, in its plan of construction, a key to the more complex structure of the higher centres. The spinal cord, as is well known, is at the same time an independent centre for reflex action, and the channel by which sensory impressions are transmitted upwards from the surface to the brain, and motor impulses downwards from the brain to the muscles. In the anterior grey cornua are seen large irregular multipolar cells, in the branching processes of which all the fibres of the anterior or motor nerve-roots find their origin. The groups of cells here found are, therefore, called the motor nerve-nuclei; only in the cord the individual nuclei are not separate and distinct, but joined into a continuous column extending the whole length of the cord—the anterior grey column.

In the posterior grey cornua, again, are found cells smaller and different in shape, in which some at least, and probably all, the fibres of the posterior or sensory nerve-roots end. These groups of cells constitute the sensory nerve-nuclei. It is impossible to demonstrate the complete termination of the posterior nerve-roots in the cells of the posterior grey columns, because many fibres turn upwards, and some downwards, before they enter the grey matter; but all the sets of fibres have by different observers been followed to cells; and when the nerve-nuclei are no longer fused together, but distinct, as in the medulla oblongata, we see the entire nerve-roots go straight

to them. Physiological reasons for the conclusion will also be given.

The mechanism for simple reflex action, then, will consist of a sensory nerve-nucleus in the posterior grey column, with its afferent nerve-root; a motor nerve-nucleus in the anterior grey column, with its nerve-fibres passing to muscles; and fibres or cell-processes connecting the two. Now, the first proposition respecting them is as follows:—

PROPOSITION I. *The same cell and fibre apparatus which serves for reflex action serves also for the transmission upwards of sensory impressions, and downwards of voluntary motor impulses.* We have not, in fact, in each posterior nerve-root, one set of fibres ending in cells in the cord for reflex action, and another which passes up to the cerebrum conveying sensation; and, again, in each anterior nerve-root, one set of fibres arising in the anterior nerve-nucleus for reflex impulses, and another set continuous with fibres in the cord which have come down from the brain to conduct volitional movements; but the very same fibres and cells in the posterior nerve-roots and nuclei receive impressions which, transmitted forwards to motor nerve-nuclei, give rise to reflex action, and upwards to the brain, give rise to sensation. The same cells also in the motor nerve-nuclei are called into action at one time by posterior nerve-nuclei in the cord, when the movements are reflex; at another, by the centres in the brain, when they are volitional. The double or multiple relation of groups of nerve-cells here indicated is a fact of great importance, and prevails throughout the nervous system.

It has long been recognized that movements require what has been called a *guiding sensation.* The structural aspect of this fact is presented by a comparison between the anterior and posterior nerve-roots and nuclei. The anterior or motor nerve-cells are large, and have numerous processes, which branch frequently in all directions. The cells of the posterior or sensory nerve-nuclei are more numerous, smaller, and have fewer processes, thus permitting more definite grouping and combination. The larger size of the posterior nerve-root, and the fact that some of its fibres pass upwards and others downwards, suggest the same conclusion; namely, that definite arrangements of receptive or sensory nerve-cells exist, which govern or direct the motor manifestations. The predominance of the sensory side of the nervous apparatus over the motor is worthy to be borne in mind.

The channel of voluntary motor impulses in the cord is found by experiment to run in the antero-lateral white column, and the decussation of the entire motor tracts of the cord proper takes

place in the medulla oblongata. The sensory channel in the cord lies within the grey matter; and the decussation takes place, not at one spot for the entire cord, but for each sensory nerve-nucleus separately just above the nucleus.

The second proposition respecting the spinal cord is the following:—

PROPOSITION II. *The spinal cord is capable of co-ordinating—i.e. of combining for orderly, purposive, or adapted action—all movements guided by cutaneous impressions.* The word co-ordination has been a kind of bugbear. From expressing a simple fact of observation it has come to mean or imply with many some special power or property analogous to volition or involving intelligence. It may be well, therefore, to explain that *co-ordination simply means the connecting together by fibres or cell-processes of cells, or nerve-nuclei, or ganglia, as the case may be, for co-operation in action.* It should be added, however, that the connexion involved in co-ordination is peculiarly intimate, and, once established, functional co-operation appears to be obligatory.

There are three chief modes of co-ordination exemplified in the human spinal cord: (1) That by which the balancing of the body in the erect position and in the act of walking is effected; (2) The bilateral association of the nerve-nuclei of muscles bilaterally associated in their action; (3) The crossed association of the anterior extremity of one side with the posterior extremity of the other side, and of different segments of the trunk.

In man, the chief spinal co-ordination is that concerned in the maintenance of the erect position and in the movements of walking. The balance of the body on the narrow base afforded by the feet is kept for us automatically by the spinal cord. We see how much better this is done than by our own conscious effort if we attempt to walk a plank thrown across an abyss. Under these circumstances, we cannot trust the cord, but must try ourselves, by the aid of vision and strict attention to our steps, to steer a right course and maintain a just poise. The result needs no description. Safety is endangered by the effort to secure it, and we go tottering and hesitating along the plank, which we should walk without the least difficulty were it lying on the ground. In this unconscious balancing action, almost all the muscles of the lower limbs and trunk take part. We tend towards one side or the other; a little extra pressure is made on the corresponding part of the feet; the impression travels up to the cord, and at once calls into action the muscles, which will correct the departure from the upright position. This involves the co-operation of many superimposed nerve-nuclei almost along the entire length of the cord, and such co-operation

implies connexion by fibres or cell-processes. The pathology of locomotor ataxy has, moreover, shown us that the fibres which effect the required co-ordination run in the posterior white columns; and they pass, no doubt, in loops, some short, some longer, from one segment of the cord to another.

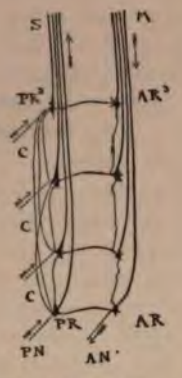

FIG. 1 illustrates Spinal Co-ordination. PR to PR³ is a series of posterior nerve-root-nuclei, with looped co-ordinating fibres (CC) of posterior columns. AR to AR³, motor nerve-nuclei. S, sensory; M, motor channel. In locomotor ataxy, the fibres (CC) being atrophied, the co-operation of different segments is prevented.

The spinal cord of the frog is generally spoken of as affording the most remarkable instance of co-ordinated or purposive reflex action. As is well known, a decapitated frog, when it has recovered from the shock of the operation, sits in a natural attitude; turns over when placed on its back; pushes away with both hind legs a straw used to irritate the cloaca, and leaps if the irritation be persistent; wipes off with the corresponding hind leg acetic acid applied to the side of the body, and, this leg being amputated, employs the other for the purpose. It has been considered that the co-ordination here seen is higher in degree than, if not different in kind from, any effected by the human spinal cord; and that, in fact, functions are delegated to the spinal cord in the frog which are exercised by the brain or sensori-motor ganglia in man. I do not accept this view. These movements in the frog are simply responses to cutaneous impressions, and far less complex and delicate than those which balance the human body. It would be much easier to construct an automaton frog performing these actions than an automatically self-balancing human figure. In my opinion, the mechanism for any movements effected through the spinal system of the frog exists potentially in the spinal cord of man;

but it is superseded by co-ordinations of higher centres, and is never called into operation; and I repudiate the idea, with all the conclusions based upon it, that there is anything higher in degree or different in kind in the endowments of the spinal cord of the lower animals.

The phenomena of *locomotor ataxy* are too well known to require description; and the loss of the power of standing erect, or of walking with the eyes shut, is at once understood by reference to the damage of the automatic apparatus. But there are other features of this condition besides the mere loss of balancing power which require explanation. Why are the legs jerked out so extravagantly and irregularly in walking? There is loss of control, it is said. But this is no answer; it is merely stating the fact in other words. We shall understand it by referring to the mechanism of the cord, as described in the first proposition. The motor impulses for the successive stages in the act of walking come down the antero-lateral columns to the nerve-nuclei of the lumbar enlargement. Normally, they would be distributed along many segments of the cord by the co-ordinating fibres in the posterior white columns; but, this dispersion of energy being prevented by damage of the tracts along which it would have travelled, the energy is concentrated on the nerve-nuclei of the lower limbs, and provokes inordinate action in them.

The precise converse of this sometimes occurs. For example, in a case under my care, there was great loss of sensation in the lower extremities, so much so that the patient, when admitted into the hospital, had large blisters on the soles of both feet, produced by a hot bottle which he had not felt. The muscles were well nourished, exceedingly powerful, and well under command while lying in bed; but the man walked with difficulty. He stood and walked, however, equally well with the eyes shut or open (a proof, by the way, were any now needed, that ataxy is not due to loss of sensation). When on his feet and attempting to walk, the muscles of the calves and thighs were found to become as hard as iron, and it was this contraction which impeded locomotion; but the most striking fact was that, if made to stand *barefoot*, the reflex action in the lower limbs was so violent as to throw him off his feet, and walking was absolutely out of the question. After putting on thick stockings and boots, he would stand and walk fairly well. Here, of course, the voluntary movements were interfered with by excessive reflex action, especially when this was intensified by contact of the naked feet with the ground; and the explanation is simple. The upward channel of sensation in the cord being damaged, impressions reaching the posterior nerve-

nuclei, which normally are divided between the anterior nerve-nuclei, where they excite reflex movements, and this sensory tract, were here concentrated on the former, with the result described. (See Diagram 1.)

I know no other explanation capable of being expressed in terms of cells and fibres, except that afforded by the mechanism I have described, which can be applied to the phenomena of ataxy and of the case just mentioned, or to the facts of inhibition, or to the exaggeration of reflex action after section of the cord.

Bilateral Association of Nerve Nuclei.—The second mode or kind of co-ordination effected by structural arrangements in the spinal cord, or its prolongation upwards as the medulla oblongata and pons, is that known as the bilateral association of nerve-nuclei. I should have been glad, had time permitted, to discuss this fully. I enunciated the hypothesis some years ago (*British and Foreign Medico-Chirurgical Review*, April, 1866). It has been of service in the elucidation of the physiology of the nervous system, and has not yet come to the end of its applications.

To explain it, I must refer to the facts of the ordinary form of hemiplegia caused by damage of the corpus striatum. In a severe case, it is observed that, while the paralysis of the arm and leg is complete, there is only partial paralysis in the face and tongue; and in the ocular muscles, the muscles of the neck, back, and abdomen, no paralysis at all can be detected. The conclusion was, that the corpus striatum could not be the motor ganglion for the entire opposite half of the body, but only for the limbs; and endless confusion arose therefrom.

But it will be seen that another interpretation is possible. When the paralysed parts in hemiplegia are compared with those not paralysed, it is found that the paralysis is complete in those muscles which are entirely independent of any muscles in the other half of the body, as is the case in the limbs. Paralysis is entirely absent, on the other hand, in muscles which never act, and cannot be made to act, without the corresponding (or some other) muscles of the opposite side. We cannot, for instance, look with one eye in one direction and the other in another, or hold one still while the other is carried round the field of vision. Now, when muscles thus constantly act together, and are incapable of acting independently, it is a physiological necessity that their nerve-nuclei in the two halves of the cord should be associated by commissural fibres, so as to become, to all intents and purposes, one nucleus. But this common nucleus will be in communication with both corpora striata; and, when one corpus striatum is damaged, it can still receive impulses from the other, so that no paralysis is produced.

This will, perhaps, be better understood by reference to a simple diagram (Diagram 2). The chains of cells B B′ represent the nerve-nuclei of the right and left brachial plexuses, not connected together by commissural fibres; B S and B′ S′, the motor channels from the corpora striata, decussating in the medulla; O and O′ represent the oculo-motor nuclei in close apposition, and intimately connected by fibres; O S and O′ S′, the motor channels from the corpora striata, decussating at once, as is the case above the common decussation of the anterior pyramids. If, now, the corpus striatum S, be destroyed, the brachial nucleus B is cut off from all volitional impulses, and is paralysed; but the oculo-motor nucleus O, though it receives no impulse from S, is reached by impulses from S′ through the fibres O′ O which join it with O′, and consequently exhibits no paralysis.

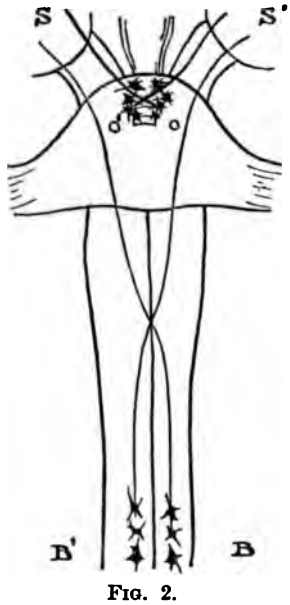

FIG. 2.

It has been put forward, as an explanation of the immunity from paralysis in hemiplegia of the muscles which escape, that their action is automatic. If this were true, it would afford none but a verbal explanation; but it is not true. No actions can be less automatic than the rotation of the head and the movements of the eyes.

It will be found that throughout the body the paralysis is proportionate to the independence of the muscles of the two halves. In

the face and tongue, where, as already stated, the paralysis is partial, the movements are habitually symmetrical, and independent movements, though possible, are extremely rare and exceptional. A minute analysis of this partial paralysis would further illustrate the general law, were there time for it. I will only mention the fact that, while a hemiplegic man can close both eyes together, he cannot, however accomplished in the trick previously, wink the eye of the paralysed side alone.

I should have been glad, again, to dwell on some of the interesting explanations afforded by the association of nerve-nuclei. One of these is of the lateral or conjugate deviation of the eyes and head observed in some cases of hemiplegia, which is usually set down as a phenomenon of irritation. The face and eyes are persistently turned away from the paralysed side, and cannot be directed towards the sound side, or brought beyond the median line. (The patient has been humorously said to be trying to look at his lesion.) The deviation is only temporary as a rule, and is due to difficulty in the establishment of the communication between the associated nuclei in a reverse direction. Sometimes, in disease of the pons, lateral deviation of the eyes is persistent.

The association of nerve-nuclei, again, affords the only explanation of the origin of the spinal division of the spinal accessory nerve so low down the cervical cord. The sterno-mastoid muscle which it supplies co-operates with the opposite inferior oblique and other muscles of the neck in the rotation of the head ; and by the filaments arising from successive segments of the cord the necessary association of the spinal accessory nucleus with the various nuclei with which it acts is accomplished.

A fact in connexion with the association of nerve-nuclei, which may be said to have established the hypothesis, is that, in hemichorea and other forms of unilateral convulsive affections, the commissural connexion of the nerve-nuclei, which prevents the paralysis of bilaterally associated muscles in hemiplegia, permits of their being reached by the irregular impulses issuing from the affected corpus striatum ; so that *hemichorea becomes bilateral exactly at those parts where in hemiplegia there is no paralysis.* Referring again to the Diagram 2, if S, instead of being destroyed, be discharging irregular impulses, they will reach B, but not B′ ; while they will reach O′ through the associated nucleus O. I was on the look out for this corollary from the moment of my enunciation of the proposition, but was anticipated in the publication of observations by Dr. Hughlings Jackson, to whose enthusiastic adoption of this hypothesis of the association of nerve-nuclei it is, indeed, greatly indebted for acceptance. We may legitimately push our

inferences further, and say that the correspondence between hemichorea and hemiplegia throws light on the pathology of chorea, and affords a strong presumption that chorea is due to an affection of the corpora striata; and other evidence in the same direction is that, in hemiplegia with spasmodic rigidity, the contraction of the paralysed muscles ceases during sleep, as do the movements of chorea, the limbs becoming flaccid, and falling into natural attitudes.

The third form of spinal co-ordination I have mentioned: the crossed association of the anterior extremity of one side with the posterior extremity of the other. This is more prominent in some of the lower animals than in man; but we see it illustrated in the swinging of the arms in walking, or especially in running. A crossed association between muscles at different heights of the two halves of the body prevails more or less, however, along the

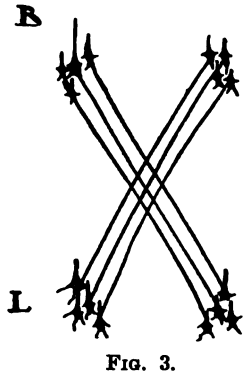

FIG. 3.

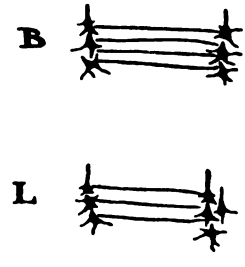

FIG. 4.

entire trunk; and I am inclined to think that this is what is represented by the decussation seen in the anterior white commissure of the cord, in which fibres from the anterior white column close to the fissure cross over to the opposite anterior grey cornu.

I refer to this crossed association, however, chiefly to show how confusion is introduced into the study of nervous physiology by the conflicting results of experiments. In the dog, which trots, the association of action is between one fore-leg and the opposite hind-leg; in the rabbit, which leaps, and never trots, the association is between the two fore-legs and the two hind-legs; and these associations are represented by commissural fibres, as seen in the two diagrams (3 and 4), in which B and L represent the brachial and lumbar nuclei. An experimenter on dogs obtains certain results; an experimenter on rabbits obtains results which differ, and at once considers himself authorized to contradict his predecessor, forgetting that the different

mode of locomotion in the two animals implies a totally different mechanism in the spinal cord.

The crossed association of the anterior and posterior extremities, and of different segments of the body, explains why an injury which in man would cause hemiplegia, in dogs usually gives rise to the *mouvement de manège*, the dog usually going round and round, as if running after his tail. Prevost of Geneva has shown that the conjugate deviation of the head and eyes in man is exactly analogous to the *mouvement de manège*; and it is interesting to note that the muscles concerned in this conjugate deviation have an oblique rather than direct association with each other; the external rectus of one eye supplied by the sixth nerve with the internal rectus supplied by the third of the other, and, as has been already mentioned, the sterno-mastoid of one side with the inferior oblique axo-atloid of the other.

The fibres, again, effecting the association under consideration are, no doubt, those which throw the hind leg into action in general convulsions, in spite of its paralysis from section of the corresponding half of the cord: a phenomenon which led Brown-Séquard to conclude that a special tract of fibres exists in the cord for convulsive purposes!

This brings up the question of multiple conducting channels in the cord, to which no reference has yet been made. It has been considered that a separate and distinct channel was necessary for every kind of sensibility and each form of motor activity: for tactile sensibility, for pain, for sensations of heat and cold, for tickling, for the muscular sense. Altogether, I think, eleven different channels are said to be required, but I do not believe a word of all this. If, in a telegraphic wire, two messages can be transmitted at the same time, it would be strange if impressions so different from each other as those enumerated could not be discriminated by differences in the molecular movement excited in the nerve-fibres and cells. We are all a little like the profane Yankee who acknowledged that man was a smart piece of work, but thought that, if he had been present at the making of him, he could have suggested "a wrinkle or two." A distinguished medical authority would make him without tonsils, and a great anatomist thinks the vermiform appendix a useless source of danger. I will not in like manner impute clumsiness to Nature in requiring a different tract and apparatus for each modification of sensation; I think it is the interpretation which is clumsy.

While, however, we need not permit ourselves to be perplexed by the complications introduced into the spinal mechanism by the

hypothesis of multiple channels for the various sensations, there is a complication which should not be lost sight of. The spinal cord is a nerve-centre for the sympathetic as well as for the cerebro-spinal system. The chain of ganglia along the bodies of the vertebrae does not constitute the spinal cord of the sympathetic system. In the early stage of development, the rudimentary cord gives off its nerves to the two primary layers of the embryo, serous and mucous alike; but, the serous layer evolving the great mass of the body, while the mucous forms only the viscera, the nerves of the latter remain small in proportion, and are represented by the inconspicuous tracts which form the roots of the sympathetic issuing with the spinal nerves from each intervertebral foramen. The reactions between the sympathetic and spinal systems, which are numerous and important, are represented by a mechanism of cells and fibres in the cord, blending with that I have just crudely sketched; but of this we know little.

The Medulla and Pons.—We come now to the medulla oblongata and pons Varolii, which are important as the seats of nerve-nuclei concerned in circulation, respiration, and other great functions, and, again, as the link between the cerebellum and spinal cord. As a part of the spinal system, the medulla is a prolongation upwards of the cord. Its nerve-nuclei are large, and distinct one from another, as befits the importance and special character of their uses; but they are not otherwise essentially different from the nerve-nuclei in the cord. There is a change in the relative position of the grey and white matter, and a rearrangement of fibres in the latter. The grey matter, from occupying the axis of the cord, gradually approaches the posterior aspect, and is spread out on this surface, forming the floor of the fourth ventricle; the central canal being laid open at the point of the calamus scriptorius. As the central canal, in successive sections from below upwards, approaches nearer and nearer to the posterior surface, the posterior grey cornua containing the sensory nerve-nuclei are gradually displaced outwards, while the anterior cornua are clubbed together at the median line; so that, in the floor of the fourth ventricle, the motor nuclei are all near the median raphé, the sensory nuclei near the margins.

Of the rearrangement of the fibres it is unnecessary to speak, except perhaps to refer to the decussation of the motor tracts in the anterior pyramids, and this chiefly in order to mention a curious instance of the way in which preconceived opinion can override observation. Under the idea that, because the motor nuclei of the hypoglossal and facial nerves are above the decussation of the pyramids, there is no crossing of the fibres connecting them

with the brain, medical men frequently state—and it was till recently so put down in some books—that in hemiplegia the paralysis of the face and tongue is on the side opposite to the paralysis of the limbs, whereas, of course, the contrary is the fact. In the medulla and pons, the communications between the different motor nerve-nuclei and the corpus striatum cross over separately, like the sensory channels everywhere.

The Cerebellum.—The functions of the cerebellum, as I understand them, I can only enunciate and briefly illustrate, without attempting to explain the grounds of my opinion, or meet the objections which might be made.

The cerebellum co-ordinates movements guided by vision, or combines the general movements of the body rendered necessary by special actions ordered by volition. For instance, to illustrate the latter function, I wish to strike a blow. I am conscious only of the desire to hit the object and to hit it hard; this is the only action realized in consciousness. But, in order to carry out the intention, not only must the fist be clenched and the arm shot out, but the feet must be firmly planted, the legs made rigid, the body thrown forward, the chest fixed; and this is what is done for me by the cerebellum.

We can see that there is no such relation between visual impressions and muscular actions as between these and tactile impressions, and any mechanism such as that for reflex response to the latter is impossible as regards vision. Visual impressions are of a different order from tactual impressions (as a cube from a square ?), compound or complex; and, as guides to movements, or to be realized in actions, require analysis, so to speak, by the intervention of a ganglion. How the cerebellum is acted upon by the cerebrum or sensori-motor ganglia, and in turn acts upon the cord, we do not yet know. We see its inferior crura, which connect it with the cord, resolving themselves into arcuate fibres, which cross to the opposite side of the medulla, and come into relation with the olivary bodies, and apparently also other grey matter. The middle crura in the pons also cross the median line, and end in nerve-cells here present; these crura probably serving to bring the cerebellum into relation with the nerve-nuclei of the medulla and pons. The superior crura pass to the "red nucleus," a large ganglion resembling the olive in structure, situate in the tegmentum of the crus cerebri; and to the sensori-motor ganglia.

The Crura Cerebri.—We come now to the crura cerebri; and the first thing to be noted is the enormous accession of fibres in the pons between the passage of the medulla under the bridge below and the emergence of the crura above. Many of these will be the

channels of impressions and impulses between the nerve-nuclei along the floor of the fourth ventricle and the sensory and motor ganglia of the brain; possibly some may arise in the cells in which the fibres of the crura cerebelli end. However this may be, it is evident that there are masses of fibres in the crus which have not come up from the cord, and which do not, consequently, belong to the motor or sensory channel of the cord.

In the crus, the sensory and motor tracts are for a time separate and distinct, and form the *tegmentum* and *crusta* respectively, the locus niger lying between them. The crusta is inferior and superficial, and is seen at the base of the brain; the tegmentum lies upon it; but, while the motor tracts (crustae) diverge, leaving between them the interpeduncular space, the sensory tracts (tegmenta) remain in apposition up to the third ventricle. Passing upwards towards the brain, the fibres of both crusta and tegmentum spread out fan-fashion, and, in doing so, change their relative position. The tegmentum, from resting upon the crusta, gets to its inner side; and the double fan formed by the two has its edges forwards and backwards. Its inner surface, formed by the tegmentum, is convex, looking inwards and upwards towards the ventricles, and has resting upon it the thalamus and intra-venticular corpus striatum (caudate nucleus). Its outer surface is concave, and looks downwards, having in contact with it the extra-ventricular corpus striatum (lenticular nucleus). The anterior edge of the tegmentum-fan overlaps the fan of the crusta, and bends completely round it near the anterior perforated space to reach the extra-ventricular corpus striatum.

The Thalamus and Corpus Striatum.—The thalamus is chiefly intra-ventricular, and rests on the inner surface of the radiating fibres of the tegmentum; but it bends round the posterior edge of the fan-like expansion of the crus as a whole at the top of the descending cornu of the ventricle, this process sending fibres backwards round the cornu to the occipital lobe, and forwards along the roof of the cornu. The thalamus exhibits, when hardened, an admixture of cells and fibres, and appears to resolve itself into masses of fibres proceeding forwards, outwards, and backwards into the hemisphere with those of the crus, those which pass forwards going beneath the intra-ventricular corpus striatum.

The corpus striatum may be said to sit astride of the anterior edge of the fan formed by the crus, and it is above as well as in front of the thalamus. The two portions of this ganglion, the intra-ventricular or caudate nucleus and the extra-ventricular or lenticular nucleus, approach each other near the anterior perforated space, but are scarcely continuous; their grey substance, however,

is continuous between the diverging fibres of the corona radiata. The grey matter is soft, and shows little intermixture of fibres.

There cannot be traced from the intra-ventricular portion any great masses of fibres passing to the hemispheres, though fibres pass round in the roof of the middle or inferior cornu of the ventricle from its posterior caudate extremity; nor can fibres of the crus be traced to it. This is probably, therefore, the less important division of the ganglion; an additional reason for this conclusion being, that it dwindles greatly in relative size during the development of the

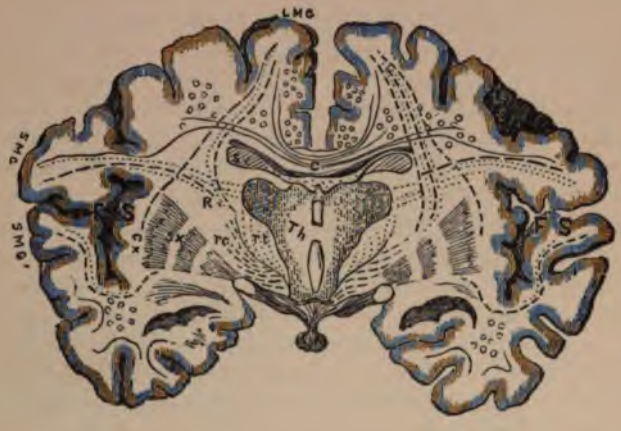

FIG. 5.—Transverse section of Brain behind Infundibulum. Sv, Intra-ven tricular; and Sx, Extra-ventricular Corpus Striatum. *Th*, Thalamus. *rc*, Crusta; and *rt*, Tegmentum of Root, or Crus Cerebri. R, Radiating expansion, or Corona Radiata. *rc*, *rt*, and R together, form what has been called the internal Capsule of the Lenticular Nucleus. Cx, External Capsule (including the Claustrum). C, Corpus Callosum. FS′, Fissure of Sylvius. LMG, Longitudinal Marginal Gyrus. SMG and SMG′, Sylvian Marginal Gyri. - - - indicate line of distribution of fibres of Corpus Striatum. . . . fibres of distribution of Thalamus. Left side of figure more accurate than right.

hemispheres. At an early period, it appears like a great roll of grey matter extending from the anterior extremity of the ventricle round the whole length of the descending cornu. Considerable mischief, again, may take place in it without giving rise to hemiplegia.

The extra-ventricular corpus striatum is encased on its outer surface, which lies immediately beneath the convolutions of the island of Reil, and on its inferior surface, by a thin layer of fibres arising in the cells of its grey matter, these fibres passing to the convolutions of the hemisphere; but most of its fibres must

issue from the part of the ganglion which is in contact with the radiating fibres of the crus, and assist to form the corona radiata. The plane of fibres limiting the grey matter of the corpus striatum externally, and separating the ganglion from the gyri operti, has been called the external capsule—a name which would be unobjectionable if it had not led to the name internal capsule being given to the mass of radiating fibres which enter the region of the central ganglia inferiorly as the crus, and emerge greatly reinforced as the corona radiata, to be distributed to the hemispheres.

The point of interest here is the relation between these sensory and motor tracts and the central ganglia, the thalamus and corpus striatum. The corpus striatum is unquestionably the motor ganglion; and I assume (though it cannot be said to be demonstrated) that all the fibres of the motor tract from the cord and medulla end in it. It will be in the extra-ventricular portion that the fibres end, for the intra-ventricular part of the ganglion is separated from the crusta by the whole thickness of the tegmentum. The thalamus I consider to be the sensory ganglion—a view not universally accepted; and I assume, again, that in it the sensory tract finds its terminus. Meynert considers that his sections of small brains of lower animals show this, as well as the termination of the motor tract in the corpus striatum. I am bound to add that, in my own dissections of the human brain, the thalamus has been easily raised from the tegmentum, as if not closely connected with it; but naked-eye dissections cannot be at all relied on to determine the relations between cells and fibres; e.g. in a cord hardened in spirit, the antero-lateral white columns can be separated from the grey columns in which their fibres end, as if they had little or no connexion with each other. There are fibres in the crus which, according to my observation, do not end in the central ganglia, but pass by them to the hemispheres;[1] but, as has been mentioned, the crus contains other fibres besides those of the sensory and motor tracts, either coming direct from the cerebellum or from cells in the pons, etc., with which cerebellar fibres are connected.

Another point of interest is the connexion between the thalamus and corpus striatum which ought to exist if they are the sensory and motor ganglia, and especially if the sensory and motor nuclei of the cord and their relations furnish any guide. The required connexion is easily demonstrated, and is found to consist of fibres issuing from the thalamus and entering both divisions of the corpus striatum. The thalamus, which, as has been said, can be raised

[1] The pyramidal motor tract has since been traced by Wallerian degeneration through the corpus striatum from the cortex to the cord direct.—Ed.

posteriorly from the tegmentum, gives off from its anterior and outer margins in relation with the intra-ventricular corpus striatum large round bundles of fibres, which follow the direction of the radiating fibres of the tegmentum. These, proceeding forwards, pass beneath the intra-ventricular corpus striatum, the soft grey matter of which dips in between the bundles.

The extra-ventricular corpus striatum is not so easily reached, both tegmentum and crusta separating it from the thalamus. Fibres, however, from the latter ganglion find their way to it round both the anterior and posterior edges of the fan-like expansion of the crus. It has already been mentioned that, anteriorly, fibres of the tegmentum turn round the edge of the crusta, to end in the external division of the corpus striatum (*rt*, Fig. 6). With these are others from the thalamus. Posteriorly, again, the thalamus curves round the crus, and ends in a tail-like process (*th*, Fig. 6), which passes forwards in the roof of the descending cornu of the lateral ventricle; its fibres forming part of the collar of the crus, and passing to the grey substance of the extra-ventricular corpus striatum beneath the uncinate lobule, as well as to convolutions.

We come now to a consideration of the function of these ganglia. The corpus striatum is the motor ganglion for the entire opposite half of the body. It translates volitions into actions, or puts in execution the commands of the intellect; that is, it selects, so to speak, the motor nerve-nuclei in the medulla and cord appropriate for the performance of the desired action, and sends down the impulses which set them in motion. These impulses are transmitted through fibres, and the fibres must start from cell-processes in the corpus striatum. *A given movement, therefore, must be represented in the corpus striatum by a group or groups of cells giving off downward processes, which become fibres of the motor tract of the cord.*

When the movement is simple, or when the co-ordination required can be effected by the cord, as in walking, the cell-group will be small, and the descending fibres few. When the movement is complex and delicate, and guided by vision or by conscious attention, as in writing or drawing, the cell-groups will be large and definite, and the descending fibres numerous. There will not be a separate group of cells for each movement; but the same cells may be differently combined, just as different combinations of carbon, hydrogen, oxygen, and nitrogen, form the basis of all organic substances. Words which require for their utterance the simultaneous co-operation of muscles of the chest, larynx, tongue, lips, etc., and the exquisite and rapid adjustment of their move-

ments concerned in phonation and articulation, must be represented in the corpus striatum by very large groups of cells, and not in that of one side only, but in both.

That the thalamus is the sensory ganglion is by no means generally admitted. Experiments have appeared to give contradictory

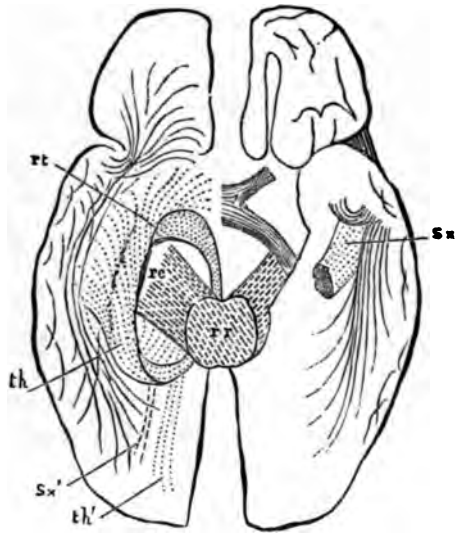

FIG. 6.—On the right side, the dissection has only been carried far enough to show the plane of fibres underlying the superficial convolutions on the inferior aspect of the Temporo-Sphenoidal Lobe, and forming the floor of the descending Cornu. This Cornu has been opened anteriorly, and fibres (*s x*) from the Extra-ventricular Corpus Striatum to the apex of the T S Lobe are seen. On the other side, the dissection has been carried much further, and the optic tract has been removed. *rr*, Crus Cerebri; *rc*, Crusta; *rt*, Fibres of Tegmentum (and from Thalamus), turning round anterior edge of Crusta; *th*, Tail of Thalamus turning round posterior edge of Crusta, forming Collar of Crus (*l'anse du pedoncle* —Gratiolet), and distributing fibres to Sylvian margin of Temporo-Sphenoidal Lobe; *th'* and *Sx'*, Fibres from Thalamus and Extra-ventricular Corpus Striatum respectively to occipital extremity of Hemisphere. The longitudinal fibres not indicated by letters belong chiefly to the System of the Gyrus Uncinatus.

results; but complete experimental destruction of the thalamus, without damage to other parts, has so often been shown to cause loss of sensation, and pathological lesions of this ganglion are in my experience so constantly associated with impaired sensation, that I cannot doubt that the thalamus holds a relation to sensation similar to that of the corpus striatum to motion. As regards

experiments which seem to show that sensation is quite independent of the thalamus, we must bear in mind that some experiments would almost seem to warrant the conclusion that sensation, motion, and intelligence were all independent of the brain.

In a paper of mine already alluded to, I attempted to meet one objection to the sensory function of the thalamus; viz. that lesion of it is not attended with impairment of sensation to the same degree that motor paralysis follows injury of the corpus striatum; and I still think the explanation there given of this fact valid. For some reason or other, sensation is more diffusely conducted than motion. If we divide the ulnar nerve, sensation is not absolutely abolished in the ring and little fingers. It seems as if there were a general network of sensory nerve-fibres at the periphery, with outlets along the different nerves. In experiments on the cord, again, it has been found that, so long as the smallest bridge of grey matter remains, sensory impressions are transmitted; and it is well known that, in disease and injury of the cord, sensation often persists when motor power is entirely lost. The thalamus then simply shares the property of the entire sensory apparatus, in giving little evidence of lesion.

The Special Senses.—Another objection I left unanswered, because the reply depended on the establishment of the hypothesis of the association of motor nerve-nuclei; and I did not wish to compromise this by putting too great a strain upon it. The objection is that, if the thalamus be the general sensory ganglion, it should serve for the special senses as well as for common sensibility; and that, when lesion of the thalamus gives rise to hemi-anaesthesia, it ought to cause also unilateral blindness and deafness, which it does not. Now, if bilateral association of muscles be accompanied with bilateral association of motor nerve-nuclei in the cord, and this prevent paralysis of such muscles in hemiplegia, bilateral combination of sensation ought to involve fusion of sensory nuclei. It is unnecessary to point out how complete is the combination of the vision of the two eyes: scarcely less complete is combination of sound reaching the two ears. There ought, then, to be such close association between the two optic nuclei and the two auditory nuclei as to make them practically a single nucleus for the two nerves sending fibres to the two thalami; so that unilateral blindness or deafness, from injury to one thalamus, would be impossible. Dr. Hughlings Jackson has lately called attention to the occurrence of hemiopia with hemi-anaesthesia due to lesion in or near the thalamus. This appears to me to be the precise analogue of conjugate deviation of the eyes.

The function of the thalamus as the sensory ganglion will be to

translate an impression arriving from the cord into a crude sensation. It is not easy to represent this in terms of cells and fibres, but it may be illustrated as follows: A finger is placed in the palm of a sleeping infant; the little hand closes gently round it. This is simply reflex. (Poor old Kentuck, in Bret Harte's story of *The Luck of Roaring Camp* would scarcely have been so excited by this simple act, had the scientific explanation of it been present to his mind.) Let the child be awake, and it will be vaguely conscious of the pressure, temperature, surface-characters, etc., of the object, but as feelings only, and without any knowledge of the external cause of its sensations. This gives roughly an idea of the office of the thalamus: it is a stepping-stone between impressions and perceptions.

An example of the automatic actions effected by the thalamus and corpus striatum (the parallels of reflex actions of nerve-nuclei) will be the involuntary shrinking from a blow and the instinctive attitude of defence. The latter is none the less automatic at the moment for being "scientific" and the product of long education and training. It is not antecedently realized in consciousness; and just as the cell and fibre apparatus in the cord concerned in standing and walking, once educated, acts automatically, so does the more complex apparatus in the sensori-motor ganglia. When a man would put up his arm, a woman might not improbably simply scream: the one and the other may be equally automatic. But we shall come to kindred subjects again.

The Corona Radiata and Course of White Fibres.—We have now to follow the fibres which issue from the region of the central ganglia to pass to the convolutions. It may be well to say, by way of recapitulation, of what they consist. We have assumed that the sensory and motor tracts of the cord and medulla have come to an end in the thalamus and corpus striatum; but my investigations lead me to conclude that there are fibres of both crusta and tegmentum, probably in direct or indirect relation with the cerebellum, which pass by these ganglia to go to the hemispheres. With these will be the fibres from the thalamus and corpus striatum; and they are all, with exceptions to be mentioned presently, so completely mixed up as to be indistinguishable. The fibres of the corpus callosum, moreover, throw themselves into the mass. The following are my conclusions as to the distribution of these fibres:—

1. Wherever fibres of crus go, thither go also fibres of thalamus and corpus striatum. These fibres of crus, thalamus, and corpus striatum, which always run in company, may be called, briefly, *radiating fibres.*

2. Wherever radiating fibres go, thither go also fibres of corpus callosum, though not necessarily in the same proportion. The course of the fibres of the corpus callosum has been variously described. They have been said to bend downwards in the corona radiata, forming thus simply a commissure between the central ganglia and crura of the two sides (Foville); or, again, to pass from the convolutions of the hemisphere of one side to the corona radiata and crus of the other (Gratiolet); but their chief distribution is as a commissure between corresponding convolutions of the two hemispheres. Accordingly, those convolutions in which radiating fibres terminate are also bilaterally associated.

3. These radiating and callosal fibres are not, as has generally been stated, distributed impartially to all the convolutions. On the contrary, many convolutions do not receive a single fibre from crus, thalamus, corpus striatum, or corpus callosum; but have only an indirect communication with the central ganglia or great commissure by means of looped fibres, which pass from them to other convolutions which are supplied with central and callosal fibres.

The statement that the fibres of the crus, thalamus, corpus striatum, and corpus callosum always go together to the same convolution, may appear to go beyond what is demonstrable, seeing that they are so mixed up as not to be traceable separately; and it is not quite what might have been expected. At certain parts, however, this is easily shown. For instance, the fibres from all these sources, which pass to the tip of the occipital lobe, form distinct masses at their point of departure, and only blend near their termination.

Again, there are certain convolutions to reach which fibres from one or other source have to take an extraordinary course. Thus, the anterior extremity of the temporo-sphenoidal lobe, and its margin which forms the lower border of the fissure of Sylvius, receive fibres directly from the adjacent corpus striatum (Fig. 6, *Sx*); the fibres of the thalamus reaching the same convolutions are given off from the process which bends round in the roof of the descending cornu of the ventricle, and spread out into the lobe (*th*); the corpus callosum is chiefly represented by the anterior commissure (in my opinion, simply a detached portion of the great transverse callosal commissure), which distributes itself along the Sylvian margin, the apex of the lobe being supplied from the corpus callosum itself by fibres crossing to it near the anterior perforated space.

Even more extraordinary is the course taken by fibres of the thalamus to reach the hippocampus major. This is in com-

munication with the corpus striatum at its uncinate extremity; with its fellow in the opposite hemisphere by the reflected part of the splenium corporis callosi, which I have called the commissure of the hippocampi; but its situation on the outer side of the great transverse fissure of the brain seems to cut it off from the thalamus. The connexion, however, is effected by the fibres of the fornix, which, as is well known, arise from the thalamus, make a figure-of-8 turn in the corpora albicantia, then take the circuit upwards and then backwards described by this body, and pass to the hippocampus in the taenia.

The convolutions to which the radiating and callosal fibres go are chiefly those along the margins of the hemisphere: the margin of the great longitudinal fissure on the one hand; the margins, superior and inferior, of the Sylvian fissure on the other, continued forwards by the inferior frontal, backwards by the inferior occipital gyri to the frontal and occipital extremities of the hemisphere respectively, which are well supplied; the free margin, again, formed by the hippocampus major. To these must be added the ascending convolutions on each side of the sulcus of Rolando, named ascending frontal and parietal, or sometimes anterior and posterior ascending parietal; and perhaps the second frontal. Callosal fibres pass more abundantly to the margin of the longitudinal fissure; radiating fibres to the Sylvian border of the hemisphere.

The convolutions which receive no fibres from central ganglia or corpus callosum are all those on the flat internal surface of the hemisphere, those on the inferior aspect of the temporo-sphenoidal lobe and orbitar lobule, the convolutions of the island of Reil, and those on the convexity of the occipital and parietal lobes not near either margin as far forwards as the ascending convolution which lies behind the sulcus of Rolando. It may seem less strange that there are convolutions without central or callosal fibres, if we recollect that nowhere do these fibres pass to the grey matter within the sulci, but only to the crests of the gyri, so that by far the greater part of the cortex is without them.

Dissection of the Brain.— To unravel the hemisphere, which may be done after careful hardening in spirit, it is best to begin on the under-surface of the temporo-sphenoidal lobe, where the superficial fibres run from each extremity towards a broad flat lobule near the centre; the deeper ones pass from the anterior extremity backwards and inwards to the inner margin along nearly the whole length, spreading out from before backwards into a beautiful plane which forms the floor of the descending cornu. (See right half of Fig. 6.) Beneath this plane are seen the fibres of the corpus callosum, anterior commissure, thalamus,

and corpus striatum, passing to the apex and Sylvian margin of the lobe; and also a longitudinal system of fibres, which converge forwards from these different convolutions to the fasciculus uncinatus, in which they pass to the orbitar lobule. This is the inferior of the three great longitudinal commissural systems, and I have named it the system of the fasciculus uncinatus.

Other fibres can be seen to converge backwards to a part of the lobe corresponding with the posterior end of the fissure of Sylvius and the part of the lateral ventricle from which the descending cornu is given off. (A pin pushed transversely inwards at the end of the Sylvian fissure would enter the ventricle at this point.) Here they bend upwards round transverse fibres from the thalamus, and then turn forwards with others from the occipital lobe in the great axial commissural system.

The fasciculus uncinatus passes under the convolutions of the inner end of the island of Reil; and, starting here, all the convolutions of the island may be removed, and can be seen to send fibres to the margin of the fissure of Sylvius, but to have no connexion whatever with the corpus striatum upon which they lie.

The arrangement of the fibres on the flat internal surface of the hemisphere is very simple. They run longitudinally from one part to another, the deepest layer starting in the quadrilateral lobule, and passing forwards round the genu of the corpus callosum. The cuneus behind the internal parieto-occipital fissure does not take part in this commissural system, but a band of fibres comes round the splenium of the corpus callosum from the gyrus uncinatus.

An interesting detail is the singular correspondence between the anatomical relations of the hippocampi major and minor. They can only have received the same name for the sake of descriptive convenience; but they share the fornix between them; they are both supplied by the reflected portion of the corpus callosum below the splenium, which may, therefore, be well called the *commissure of the hippocampi*; and they are connected together by fibres which come from the grey matter in the groove of the hippocampus major over the edge of the gyrus uncinatus, twisting round each other like a rope at this point, and then spreading out again in the wall of the calcarine fissure.

The most complicated part of the hemisphere is that forming the convexity of the parieto-frontal lobes; and only an outline of the structure can be given. It has been already stated that the callosal and radiating fibres meet and partly mix along the upper and outer edge of the central ganglia. From this axis, a plane of fibres, chiefly callosal, passes upwards to the margin of the longitudinal fissure; another, chiefly radiating, horizontally outwards to

the Sylvian border; and in the angle runs the great axial longitudinal commissure, containing fibres which pass from the temporo-sphenoidal and occipital lobes forwards to the frontal and parietal lobes, and also between intermediate convolutions. In the parietal lobe there are also masses of fibres having a more or less transverse direction: for instance, between the postero-parietal and supra-marginal lobules.

Farther forwards, the central fibres are not distributed exclusively to the margins, but along the entire length of the ascending convolutions which run from the Sylvian margin of the hemisphere to the longitudinal on each side of the sulcus of Rolando. This is very difficult to make out, because not only are there fibres of the axial commissural system to these gyri, but proper fibres running in the direction of their length. I have, however, seen it strikingly demonstrated (as I have most of the main facts here stated) by the natural dissection performed by extravasated blood. The second frontal gyrus, though not marginal, appears also to receive central fibres.

We can better understand the absence of symptoms when the cerebral hemisphere is excavated by an abscess or destroyed by a tumour, when we know that this may take place without affecting fibres passing between the crura or sensori-motor ganglia and the convolutions, and shall ultimately find it less necessary to resort to the assumption that there is a vast surplusage of brain. We shall also, I trust, be less vague in our ideas of compensation or *suppléance*, and know as definitely the cell and fibre mechanism by which the function of a damaged part of a nerve-centre is replaced, as we know the collateral circulation by which we have compensation or *suppléance* when an artery is occluded.

The Evolution of Thought. — The functional mechanism of the cerebral hemispheres is at once suggested by the facts of its anatomy, and so obviously, that it may, perhaps, be well to say that the anatomical investigations preceded entirely the physiological deductions. The convolutions which have no direct connexion with the crus and central ganglia, and which are thus withdrawn from immediate relation with the outer world, will clearly be those concerned in the purely intellectual operations which can be carried on independently of existing sensations, and without at once prompting movements. They will, on the one hand, receive the raw material of thought from convolutions in relation with the sensory ganglia and tracts, and on the other will employ convolutions in communication with the motor ganglia and tracts to transmit to the muscles the volitions which are the product or outcome of thought.

Now, the convolutions pointed out by structural arrangement as the seat of the higher intellectual operations are those which are gradually superadded to the fundamental convolutions in the progress of development, which of itself is a strong reason for the same conclusion. They are, moreover, those which distinguish the human from the simian brain. The motor centres in the convolutions, again, identified by the experimental researches of Hitzig and Ferrier, and Ferrier's recently discovered perceptive centres are in situations in communication by central fibres with the sensory and motor ganglia and tracts, while the intermediate convolutions are not irritable.

The following, then, is in outline the theory of the employment of the structural arrangements of the brain in the evolution and expression of thought. In the thalamus, as has been already stated, we assume that impressions undergo the first development into crude sensations. These sensations will be transmitted to the hemispheres by fibres radiating from the thalamus to some part of the marginal convolutions, in which a further development is effected translating or transmuting sensations into perceptions. Perception is the conscious recognition of the external cause of a given sensation; is objective, and not merely subjective; and it is a necessary postulate, that each kind of perception will have its own special seat. And Dr. Ferrier has recently identified the position of the chief "perceptive centres." The visual perceptive centre is found in the angular gyrus round the end of the fissure of Sylvius; the auditory is situated near the apex of the temporo-sphenoidal lobe.

Now, ideas result from the combination of perceptions derived from vision, touch, smell, taste; and the structural counterpart of this operation will be the convergence of fibres from the different perceptive centres to a common spot, which will be in one of the superadded convolutions. But an idea is something beyond the mere combination of perceptions; there is an intellectual elaboration and the association of a name which is thenceforward the symbol of the idea, recalling more or less vividly the whole group of perceptions it represents. Names or ideas then become the subjects of thought, or material for intellectual operations.

We can already imagine breaches in different parts of the nervous mechanism, and it may be well to consider the effect before going further. For instance, we can, in the case of vision, imagine a lesion between the thalamus (assumed to be the seat of the crude sensation of sight) and the visual perceptive centre. The subject of such a lesion would see an object, but not recognize it. This seems to have been the case with Professor Ferrier's monkey, in which the visual centre of one hemisphere had been excised;

but the condition was only temporary, as might be anticipated from the bilateral association of the visual nerve-nuclei.

Again, we can imagine *a lesion to the fibres passing from the visual perceptive centre to the naming centre*, in which perceptions are combined and elevated into an idea to which is affixed a name. Under these circumstances, an object would be recognized, but could not be named. An interesting case which came under my observation exemplified this condition, and at the same time confirmed Ferrier's experimental localization of the visual perceptive centre.

The patient, a gas-inspector, of remarkable energy and intelligence, after an acute cerebral attack had entirely lost the power of naming objects at sight, and of reading. He talked fluently and intelligently, scarcely ever made a mistake in words, but was sometimes at a loss for a name, especially of a street, place, or person. He was, however, quite unable to read, or even to name a single letter; the only exception being, that he recognized his own name, whether written or printed; though even here he did not know whether the Christian name or initials only were given. While this was the case, he wrote correctly from dictation, and took notes of my instructions, which he could not read a moment afterwards; he explained that he was forgetful, and his wife would make them out. If a hand, or an article of clothing, or any familiar object, were shown him, he was quite unable to name it; while, if the name came up in conversation, he spoke it without hesitation. Asked the colour of a card, he could not give it. "Is it blue?" "No." "Green?" "No." "Red?" "Well, that's more like." "Orange?" "Yes, orange." A square and a circle were drawn, and he was asked to name either. He could not do it; but, when the circle was called a square, he said, "No, but that is," pointing to the proper figure.

It was evident that we had here exactly the condition supposed. The act of perception was perfect, but a breach had been made in the communication between the visual perceptive centre and the centre in which the sight of an object called up the name. The patient died from apoplexy; and the recent haemorrhage, having started in the old lesion, made the precise localization of this difficult; but it was clearly made out to be situate in the white substance lying between the posterior end of the fissure of Sylvius and that part of the ventricle from which the descending cornu starts. It would, therefore, isolate more or less completely the angular gyrus in which Professor Ferrier places the visual perceptive centre.

While this case is fresh in the memory, we may try to imagine

what would be the effect of a corresponding *lesion between the auditory perceptive centre and the naming centre.* It would be exceedingly perplexing; for not only would words addressed to the subject of the lesion fail to revive the appropriate idea, but the patient would not from the sounds he uttered know what he himself was saying; he would have no check on his own words, and might or might not say what he intended. I have seen cases which I consider to have been of this sort, the patient not understanding even a simple request to put out the tongue or give the hand, but responding immediately to signs, in one instance giving distinct but quite irrelevant answers, in another talking inarticulate gibberish. Cases evidently of this kind have been described as examples of aphasia, thus confounding together conditions altogether different, and introducing confusion into the question of the mechanism of speech.

We have followed the mechanism of the intellectual operations up to the act of "naming," which is the starting point of all the higher mental processes. These we cannot now trace further; but, taking for granted for the moment the conclusions formulated, that the perceptive centres are situated in the marginal convolutions, receiving radiating fibres from the thalamus, and the ideational centres in the superadded convolutions, which are not in direct relation with the central ganglia, we have this further conclusion, that the perceptive centres are bilaterally associated by the corpus callosum, while the ideational or intellectual convolutions are independent of those in the opposite hemisphere. We can thus see an anatomical possibility of the education of one hemisphere only for intellectual expression revealed by aphasia. This is, of course, assuming the accuracy of my statement that callosal fibres go to the same convolutions as radiating fibres, and there only. Of this I am confident; as also of the unexpected and unexplained fact that fibres from corpus striatum and fibres from thalamus are invariably found going to the same part, so that there is no structural indication of separate sensory and motor or receptive and emissive areas in the hemisphere; nothing to suggest which is the "way in," and which the way "out."

I assume, as will have been obvious, that we think in remembered impressions, no mention having been made of motor intuitions as elements of thought. Perhaps this is because certain heights of speculation are beyond my reach, or some orders of ideas too original for my appreciation. The idea has often occurred to me that, just as vibrations may become too rapid to give rise to audible sounds, and as the limits of audible vibrations differ in different individuals, so that a painfully shrill sound to one may be dead silence to another,

so minds are differently organized, and speculations of absorbing interest to one are meaningless to another. But, whatever the reason may be, I have not attached great importance to the motor element in thought; and, indeed, throughout the nervous system, the sensory or receptive side of the apparatus has appeared to me to have the predominance, as is illustrated in the mechanism of the cord. Were remembered motions important factors in thought, we could scarcely have ordinary intelligence in the subject of congenital chorea, incapable through life of any precise movement, or anything but idiocy in congenital aphasia; and examples are seen in which the reverse is the case.

We come now to the transmission outwards of the products or results of intellectual operations—the initiation of voluntary actions. The first step in this is the realization in consciousness of the action to be performed, which, I suppose, will take place in the superadded convolutions, and will be in effect a part of the mental operation. The ideal movement will then be analysed, so to speak, and its component elements referred to the special marginal or other convolutions in relation with the corpus striatum appropriate for their performance: the convolutions identified by Hitzig and Ferrier as the motor centres of the hemisphere. We have here a corresponding operation to the synthesis of ideas on the sensory side. Lastly, by these the cell-groups of the corpus striatum will be put in action; and we thus return to the cord.

This simple outline gives, however, a very inadequate idea of the mechanism of the process. To understand this even slightly, we must go back. We have seen that movements will be represented in the corpus striatum by a group of cells joined together by processes, and sending down fibres to the cord. These groups have to be formed; certain combinations of cells have to be selected out of an infinite number of possible combinations, as best adapted for the particular purpose. Such grouping is the structural aspect of education, so far as the corpora striata are concerned.

The Process of Education of the Nerve Centres.—We may trace briefly the process of education of the different nerve-centres, expressed in terms of cells and fibres. Beginning with the cord, its proper education is exemplified in the process of learning to stand and walk. These acts, when once fully acquired, become almost purely automatic, and are carried on by the cells and fibres of the cord, with only just the degree of direction and control from higher centres requisite to make them available for employment. The education of the cord for the purpose of walking, then, will consist in the formation of commissural connexions between different groups of sensory and motor cells (i.e. of posterior and anterior nerve-

nuclei in different segments of the cord) so intimate that the relation between these groups becomes almost as close as that between the groups of cells concerned in the elementary reflex actions of sucking, winking, etc. It must be noted, that the formation of these commissural relations takes place at the instigation of a higher centre, and under its direction and control. The child tries to stand and walk, in imitation of older people, and it pays attention to the act, in order to avoid falling. Once, however, it has learnt to walk, it does so not for the sake of walking but to go from place to place; and ceases altogether to pay attention to the act of walking. That is, when the spinal cell and fibre apparatus is arranged, it is afterwards set in motion like a machine.

In the education of the hands to precise movements, the part taken by the higher centres is far more considerable, and less is left to spinal co-ordination. Still the grouping of cells in the cord is an essential condition of the process, and the posterior nerve-nuclei are an indispensable part of the mechanism. The case will be more simple if we suppose the child to be blind, so that the visual direction of movements has not to be taken into account. It is then obvious that the guiding sensation which is necessary for precision must be tactual, and must, therefore, come through the posterior nerve-roots. The cell-processes, in fact, which connect together the cells of the posterior and anterior cornua, serve not only as channels for impressions which give rise to reflex actions, but in forming the definite cell-groups which represent definite movements.

But, while the nerve-nuclei in the cord are being connected, a far more intricate and complex grouping of cells and fibres is taking place in the sensori-motor ganglia and cerebellum, on the perfection of which will depend the harmony and grace, agility and precision, of the movements generally. To recur to an illustration already employed, a threatened blow will, in an untrained nervous system, give rise to a vague and useless start; while, after practice and education, an attitude of efficient defence would be assumed automatically. If, in boxing or fencing, the impression of the adversary's blow or thrust had to reach the hemispheres, and there excite a conscious mental representation of the counter-movement, the action itself would come too late. This representation in consciousness occurs in acquiring the respective accomplishments; but the completed education is represented by the organization of cell-groups in the corpus striatum, which can be brought into action on the arrival of certain impressions in the thalamus, without reference to the hemispheres for guidance.

Another kind of education altogether is, moreover, going on from

early childhood. The ganglia of crude sensation, the perceptive centres, and the ideational or naming centres, are successively brought into operation; but impressions arriving at the higher centres no longer result in instant outward impulses determining motions; they are stored up (fixed, perhaps, by chemical changes in the nerve-cells, somewhat as luminous vibrations by leaves). On the store of perceptions, their number, variety, accuracy, definite character, and orderly arrangement, will depend greatly the future mental character; that is, associations are formed between numerous groups of receptive cells, instead of between receptive and emissive groups.

Now, perceptions must be objective, to be real; must come through the senses. Names or ideas symbolize facts and correspond with the outer world, only so far as they represent real perceptions; and reading can only supply ideas based upon or derived from the pre-existing ideas which have been formed through the senses. The inference is obvious. The only solid foundation of what is generally meant by education is education of the senses, the perceptions (observing faculties), and limbs, by contact with external nature, after which, and only then, the child will be in a position to profit by books. In a town-reared child, impressions tend to rapid outward expression, which is incompatible with fixation and storing up of real perceptions. There is thus early quickness, and apparently greater intelligence, when compared with a country child; but no capital of sterling perceptions has been laid up. The automatic apparatus has been cultivated at the expense of the intellectual, and later the position is reversed.

Mechanism of Speech.—I may now resume my observations on the mechanism of speech, and with these conclude. From one aspect, speech is coextensive with mind. Language, spoken or unspoken, is the great product of the intellectual operations. Ideas are formed, associated, and compared; new ideas evolved, etc. The operations are still concerned mainly with remembered impressions, though symbolized by words. When ideas seek expression, a different set of operations comes into action. We know well that there is no necessary relation between the reflective and executive or expressive faculties; that one or other often predominate greatly; that, in fact, such predominance gives a character to the head and features. The turning point at which a mental tends to become a motor act may be said to be the mental rehearsal of a phrase. This we have assumed to take place in superadded convolutions; and an analysis of the ideal movement now refers its constituent elements to the appropriate marginal convolutions to be put in execution. Pathology has taught us that the way out for words lies through the

third frontal gyrus, and, very much to our astonishment, the third frontal gyrus of the left hemisphere only. This I consider to be established; and also that the explanation of it is, that the left hemisphere alone is educated for intellectual expression.

From another point of view, words, the vehicles of thought, are mere motor processes, and may be considered as if entirely devoid of significance. They may never have any intellectual significance as when learnt by a parrot; or they may survive, as motor processes, their significance as mental symbols in some forms of insanity and in some derangements of speech. In this aspect—that of mere movements—words will be represented, as already mentioned, by groups of cells in the corpora striata; and the formation of these groups furnishes another illustration of education.

Just as a child, in its early attempts to walk, is stimulated thereto by a desire to imitate those around it, and attends to the act itself till the commissural connexions between different cell-groups which constitute the automatic apparatus for walking are brought into operation, so, in its first attempts to talk, the child is influenced by imitation and guided by the ear; that is, as the grouping of the motor cells in the cord is effected through the sensory cells by cell-processes passing from the posterior to the anterior nerve-nuclei, so the grouping of the cells in the corpus striatum will be effected through the cells of the auditory perceptive centre by means of fibres connecting together the two: an explanation, perhaps, of the fact that fibres from the corpus striatum and thalamus pass to all marginal convolutions, whether in the motor or sensory regions, of the hemispheres. And, as the motor nuclei of the cord can still be employed in reflex action through the sensory nuclei, as well as in voluntary motion by means of descending fibres from the corpus striatum, so may the word-groups in the corpus striatum be reached imitatively through the auditory perceptive centre, as well as through the third frontal gyrus.

We come now to an important consideration. The auditory sense centres and perceptive centres concerned in the formation of the word-groups being bilaterally associated, and the motor nerve-nuclei concerned in the utterance of words—those of the chest, larynx, tongue, and lips—being also bilaterally associated, the word-groups must be formed in both corpora striata, although the left hemisphere only is educated for speech. Were it otherwise, and were word-groups formed in the left corpus striatum only, we should have aphasia equally from damage to this ganglion as from lesion of the third frontal gyrus of this side, which is not the case. Right hemiplegia is common enough without aphasia, and I know of no case on record in which lesion of the corpus striatum not extending

higher has given rise to loss of speech. When the left corpus striatum is destroyed, the direct way out for words being destroyed, a way round is taken, starting from the third left frontal to the third right frontal gyrus, passing thence to the word-groups in the corresponding corpus striatum; thence, of course, to the bilaterally associated nerve nuclei in the pons, medulla, and cord.

This, together with one or two other points, will be better understood by reference to the accompanying diagram, in which the parts concerned are indicated by their respective initials. The left third frontal gyrus (L. 3rd F.) being considered as the instrument of the brain for speech or intellectual expression, loss of speech, or aphasia may be produced (*a*) by destruction of its grey cortex; (*b*) by its isolation through damage involving, say at r. c., the fibres connecting it with the corpus striatum and those crossing in the corpus callosum (C. C.) to the corresponding gyrus on the right side (R. 3rd

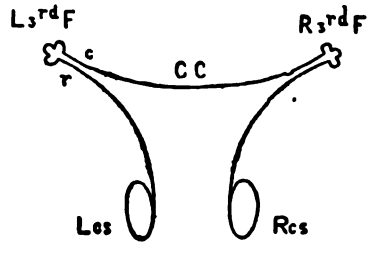

FIG. 7.

F.); this is, I believe, what happens in some, at any rate, of the cases in which aphasia is produced by lesion of the left island of Reil, the damage penetrating deeper than the convolutions of the island; (*c*) by simultaneous lesion of the left corpus striatum (L. C. S.), and of the corpus callosum (C. C.)

The left corpus striatum (L. C. S.) being destroyed, aphasia is avoided, as stated above, by speech crossing from L. 3rd F. by C. C. to R. 3rd F., and descending thence to R. C. S. and the nerve-nuclei. The difficulty in taking this new route explains the great temporary embarrassment of speech often met with in right hemiplegia. It should not be understood that the corpus callosum, right third frontal gyrus, and corpus striatum are habitually passive. I should conclude that they are normally employed in every act of speaking; my idea of the co-ordination of centres on the same level being, that co-operation is obligatory; that, in fact, centres so connected hold each other in mutual tension, discharge of one

implying discharge of the other. A higher centre controls altogether a lower centre, and discharges or inhibits it, so to speak, at will. Stammering I suppose to be due to want of accord between the discharges of the two corpora striata, so that the bilaterally associated nerve-nuclei in communication with both receive discordant impulses; and not to want of accord between the muscles of phonation and those of articulation.

There are still many points of great interest which ought to have been considered. It has been difficult, indeed, to decide what should be discussed and what passed over. Fortunately, there are limits of time and space, or, in the attempt to make this lecture comprehensive, I might have made it tedious.

ON A CASE OF AMNESIA WITH POST-MORTEM EXAMINATION

Medico-Chirurgical Transactions, 1878

In Vol. LV. of the Society's *Transactions* for the year 1872 there is a paper by the writer on the "Mechanism of Speech and Thought," some of the hypothetical conclusions of which it will be necessary to restate very briefly, in order that the bearing of the case, which forms the subject of this communication on the question of the cell and fibre apparatus concerned in intellectual operations may be understood.

Dr. Bastian's hypothesis was accepted, which assumes that there will be a certain district or area of the surface of the cerebral hemisphere in relation with each sense, in which the sensory impression undergoes the intellectual elaboration by which it is translated, so to speak, into a perception or recognition of the external cause of the sensation. These are called "perceptive centres." At that time nothing was known of their situation, but investigations into the distribution of the fibres passing from the crus and central ganglia in the "internal capsule" and "corona radiata" to the convolutions, had led to the inference that these "perceptive centres" would be found in certain convolutions in which the radiating fibres terminated. Ferrier and Hitzig have since identified these "perceptive centres," and the former, experimenting on a monkey, localizes the perceptive visual centre in the angular gyrus, the auditory centre in the inframarginal gyrus of the temporo-sphenoidal lobe, smell and taste in or near the uncinate lobule, touch in the uncinate gyrus. These are all in the marginal convolutions, which anatomical research had indicated as receiving radiating fibres, and as being thus in direct relation with the outer world, either receptively through the senses or through the muscles. These particular convolutions, moreover, all receive their fibres from that part of the thalamus which may be called extra-ventricular, namely, the process which bends round the posterior edge of the internal capsule.

It having been found that certain other convolutions were not directly connected with the central ganglia or crus, but only

with other convolutions by arcuate fibres, the speculation was pursued further. It appeared probable that the association together of the different perceptions of a given external object, which would by their combination constitute the complete idea of the object, would be represented structurally by the convergence of arcuate fibres from each of the perceptive centres to some of these "superadded" convolutions. Since, moreover, the fully formed idea implies the attaching of a "name," the convolution in which the perceptions were combined into an idea would also be the *naming* centre, and would be concerned with *words* as auditory impressions and intellectual symbols.

It was not attempted to follow the mechanism of thought further than this rudimentary step, but the study of words was taken up from the opposite aspect as motor processes. With this, however, the present communication is not concerned.

On the above view of the structural arrangements for the formation of an idea, it was pointed out as a possible occurrence that the fibres of communication from one or other of the "perceptive centres" to the "naming centre" might be destroyed by disease. Supposing this to occur in the case of the *visual* perceptive centre the sight of an object would then no longer recall the name. This, condition was actually illustrated by a remarkable case.

A very intelligent man, who talked fluently and well on any subject, and who wrote correctly, spontaneously, or from dictation had completely lost the power of naming anything shown to him. He could not after prolonged and painful effort name so familiar an object as a hand, or coat, or table, or recognize a letter of the alphabet, or read a word even of what he had himself written a moment before. The lesion giving rise to this condition was situated in the white substance of the left hemisphere, between the posterior extremity of the fissure of Sylvius and the lateral ventricle, where this gives off the descending cornu.

With this illustration in mind let it be supposed that there had occurred a similar rupture of the communication between the *auditory* perceptive centre and the *name* centre, or *idea* centre, as it may indifferently be called. It will be seen that the effects will be of a far more complex character. If a spoken word conveyed no more idea to the mind than the written word in the case alluded to the subject of the lesion would understand absolutely nothing that was said to him ; a more serious and perplexing difficulty, however, would be that he would not know what he himself was saying, his own speech would be as unintelligible to him as his own writing was to the patient whose case has been quoted. This condition was supposed to be illustrated in Case 9 of the former communica-

tion, but the patient was seen only once in consultation, and no post-mortem examination was permitted.

Another condition may be imagined, that is, *lesion in the name or idea centre* itself, and this is what is believed to be illustrated by the case now to be related.

James S——, aet 60, an omnibus driver, was admitted into St. Mary's Hospital on October 16, 1877. He had been a fairly healthy man, and was described as having been particularly intelligent, a good talker, reading much, and able to write well. He had been addicted to drink in the form of heavy bouts from time to time with sober intervals. In May, 1877, he lost his wife, and had been drinking hard, with little intermission, ever since.

A fortnight before his admission he had some sort of fit, of which no reliable description could be obtained, and from that time had been unable to speak intelligibly, and had kept his bed.

On admission he was well nourished, had a good colour, a dark complexion, and curly dark hair turning grey. The expression of his face was rather anxious and inquiring.

He walked into the hospital and up the stairs to the ward guided and assisted by friends, and had free use of both upper extremities. There was thus no hemiplegia, but slight paresis of the right side of the face was observed. Later, it was discovered by the clinical clerk, Mr. Handfield Jones, from whose careful and able notes the account of the case is abstracted, that there was decided impairment of sensibility in the right half of the body.

The most striking feature in the case was that the speech was for the most part an inarticulate jargon. When questions were asked he replied, but as a rule nothing at all resembling a word could be detected in what he said. The voice was inflected naturally, and he appeared to think that he was understood. Besides answering questions he would go on talking, addressing himself to one or another of those present, the whole being an incomprehensible ramble, but at times a distinct word or phrase would slip out. He was emotional and often cried, and it was not difficult to make out that he was begging for something—probably drink; the word rum was distinguished once or twice, and he would become excited as he went on and end by crying. When excited the phrase "If you please" was several times heard distinctly.

It was difficult to make out how far he understood what was said to him, his answers being unintelligible. He turned over in bed when told, but probably he was assisted by signs and by assistance offered him. When questioned about his symptoms he did not appear to comprehend at all what was asked. He was, moreover, obviously quite unconscious of the fact that his own speech was

gibberish, and appeared to have some idea in his mind and to think that he was giving expression to it.

The pulse rate was 140, but many beats failed to reach the wrist. There was no cardiac impulse anywhere. The heart sounds were feeble; and near the apex the first was short and sharp, and either it was reduplicated or there was a slight presystolic murmur; the second sound was inaudible at the apex. The breathing was hurried and jerky. Urine high coloured, acid, sp. gr. 1021, containing no albumen or sugar.

He was put on simple diet with milk and beef tea. An aperient was ordered, and carbonate of ammonia 4 grains and tincture of digitalis 10 minims were given every four hours. His general condition improved rapidly.

On the 19th the pulse was 120, weak, soft and regular; the respirations 40.

On the 22nd. Pulse 96, soft, but more full and slightly irregular; respirations, 28.

On November 3, the pulse had fallen to 60.

My own interviews with the patient were so perfectly similar that the indications obtained may be summarised.

The speech had the same peculiarities throughout. When asked a question he would make a brief reply as if he understood and answered; the modulation of the voice and the emphasis were perfectly natural, and corresponded with the facial expression and gestures, but as a rule there was not the least semblance to words in what he said. He never even gave a simple yes or no. He would often address a long story to us, sometimes argumentative in tone, with much appropriate gesture and corresponding changes of facial expression; or he would obviously be making some request, and would plead with great earnestness, going on to crying and beseeching. We sometimes guessed that he wanted alcohol, and once he apparently wished to leave the hospital. It was when he was excited and emotional that words and phrases would slip out in the torrent of inarticulate sounds, such as "If you please," "Thank you, thank you."

It was a matter of great interest and importance in the estimation of the mental condition of the patient, and of the loss of intellectual faculties he had sustained, to make out how far he understood what was said to him. His replies were often so suitable in length and emphasis that had there been no means of forming a judgment it must have been supposed that he had comprehended the question. When, however, he was told to do anything it was seen that he did not understand the simplest phrase. He sat up in bed once or twice when required to do so, but as this was not made a test ques-

tion, there would be other indications of what was wanted, and he was extremely ready in comprehending signs. When told to give his hand he invariably put out the tongue, and would do this several times during the same visit. When told to shut his eyes he sometimes obeyed and sometimes did not, but whether he closed the eyes or not, he put out his tongue.

The direct question whether he would like any of the intoxicating drinks he was known to be fond of was avoided, as there was no intention of allowing him any, and it would have been cruel to tantalize him, but when they were mentioned in his presence, or when allusion was made to his habits, there was no indication that he understood what was said; the opportunity was not seized of manifesting his desire when the name was mentioned, as in ordinary aphasia. On one occasion only, when the sister asked him what he had had for tea, he is said to have answered "Bread and butter."

Other interesting particulars were obtained by Mr. M. Handfield Jones. It has already been stated that sensation was found by him to be impaired in the right half of the body and limbs. At almost every visit the patient implored him for something, and the work "Drink" could sometimes be made out; "If you please," "Thank you," also were distinguished; he frequently cried, but less after he had been in the hospital for ten or fourteen days than at first.

Mr. Jones one day handed him a letter addressed to him at the hospital; he took it, appeared to read the name and address, and then put it down. Not attempting to open it, Mr. Jones then wrote on a piece of paper "Give me your hand," and called the patient's attention to it; he took it, held it so as to get a good light on it, and then, having apparently read it, laid it aside without giving his hand, though asked to do so by word of mouth as well as in writing.

He lay in bed and manifested no desire to rise. He would sit up in bed and appear to watch with interest what was going on in the ward, but did not seek to enter into conversation with other patients. When the meals were brought in he looked for his, and ate his food naturally. When the bowels were about to act he called the attention of the nurse by knocking on his locker, and then pointed to the commode. There was never anything extraordinary in his behaviour.

On November 3 he was got out of bed and dressed. He walked with rather a tottering gait to the fire and sat there for a short time; after which he rose from the chair, walked back to bed, and got in with his clothes on.

His death was sudden and unexpected. During the night,

November 5 and 6, the resident medical officer was called to him. Before his arrival the patient had got out of bed and taken a large dose of his medicine (about 30 minims of tinct. digitalis); he had then tried to reach the stool, but could not. He was found in a state of extreme dyspnoea, and blue and livid in the face. Artificial respiration was tried, but he died in a very short time asphyxiated.

Post-mortem examination.—The head only was allowed to be examined. When the skull-cap and dura mater, which presented nothing remarkable, had been removed, the pia mater was seen to be white and opaque over the convexity of the hemispheres generally, and to be raised from the convolutions by a considerable amount of fluid. This gave an opalescent appearance to the convexity of the brain. On careful examination a yellow discoloration could be seen through the membranes and fluid at the situation of the postero-parietal lobule near the longitudinal fissure, and could be traced downwards towards the posterior end of the fissure of Sylvius. This part was also soft to the touch.

When the brain was removed from the skull it was found to weigh 47 oz. The vessels at the base were almost free from disease. The membranes here were transparent, and when the fissures of Sylvius were explored nothing abnormal was detected till the extreme end of that on the left side was reached, and here the middle cerebral artery was found to be occluded by a fibrinous plug.

The two hemispheres being separated it was seen that the ventricles were in a slight degree distended by clear fluid, but all the parts seen in them were normal, and the septum lucidum intact.

The right hemisphere, except that the membranes on the convexity were opaque, and that there had been fluid beneath them, was sound.

The left hemisphere presented obvious disease, limited to its posterior half. The frontal and ascending parietal convolutions and the anterior part of the temporo-sphenoidal lobe were healthy in appearance and consistence.

Above the posterior end of the fissure of Sylvius the convolution forming the supramarginal lobule was yellow in colour, shrunken in volume, and soft. This condition extended upwards and backwards to within about half an inch of the longitudinal fissure just in front of the external parieto-occipital fissure, involving, therefore, the postero-parietal lobule. Extending backwards the morbid change implicated the angular gyrus, and nearly reached the occipital lobe; in a downward direction the adjacent parts of the temporo-sphenoidal lobe, the posterior end of the inframarginal, and parallel gyri, were soft, but not wasted or discoloured on the surface.

1. Examined by a succession of vertical transverse sections from the frontal extremity of the hemisphere backwards, softening was first encountered in the lower end or foot of the posterior ascending parietal convolution, quite at its posterior part; it did not here extend into the fissure of Sylvius, and there was no external discoloration. On the lower side of the fissure in the temporo-sphenoidal lobe, there was softening in the inframarginal gyrus to the depth of half an inch.

2. Half an inch further back, and about three quarters of an inch from the end of the fissure of Sylvius, the section found the substance of the supramarginal lobule soft and almost diffluent, and its surface in the fissure of a brownish-yellow colour; the temporo-sphenoidal lobe was also softened nearly to its inferior surface.

3. A section across the extremity of the Sylvian fissure passed through the maximum of softening, which here extended from near the longitudinal fissure, across the convexity of the hemisphere, to within half an inch of the inner and inferior border of the temporo-sphenoidal lobe; the greatest depth of the softening was midway and measured about three-quarters of an inch. In successive sections behind this point, the area of softening became narrower and its depth less; it extended backwards midway between the upper and lower borders to within an inch of the occipital extremity, being very shallow at its posterior part. It involved of course the angular gyrus and the middle *pli de passage*. The softening did not at any part reach the central ganglia or the ventricle.

The Lesion.—The part of the hemisphere affected was thus the regions supplied by the third and fourth branches of the middle cerebral artery, and the convolutions softened were the postero-parietal lobule, incompletely and superficially; the supramarginal lobule throughout its extent, and in depth down to the oval white centre; the entire angular gyrus profoundly; the annectent gyrus behind it superficially; the posterior half of the inframarginal or first temporo-sphenoidal gyrus; and opposite the termination of the fissure of Sylvius almost the entire thickness of the temporo-sphenoidal lobe. The frontal lobe and the oblique ascending parietal convolutions, in which the motor centres have been localized, were, as has already said, free from disease.

Whatever may ultimately be the interpretation placed upon them, cases like this are worthy of careful study. A definite lesion is found, to which, from the history of the attack, there can be no hesitation in assigning the symptoms. It is important, therefore, that a definite idea should be formed as to the nature and extent of the loss of function sustained. Under each of the general descriptive terms in common use, such as coma, delirium, uncon-

sciousness, insensibility, are included conditions essentially different, and an analysis of them may be as fruitful of results as a "study of convulsions" has proved to be in the hands of Dr. Hughlings Jackson. Even words like aphasia and amnesia, brought into use for the express purpose of defining a single invariable condition, become snares and hindrances by being employed to designate by the same name affections which a careful description would have shown to be different.

Commentary.—The patient in the case under consideration had lost the faculty of understanding words spoken or written by himself or others, so much so that he was not even aware that his own attempts to talk were simple gibberish; the question is whether this alone would account for this condition, or whether there was not some further and more general impairment of the mental powers. It was of course impossible to test his memory, but he recognized his friends and knew what he might expect from his medical attendants and from the nurses, addressing to the former his inarticulate requests, and summoning the latter when he required their assistance. He manifested great disappointment also when any resident medical officer or clerk passed through the ward without noticing him.

Again, he had obviously ideas in his own mind to which he thought he was giving expression, and his gestures and changes of facial expression were remarkably significant and appropriate. On superficial observation he might have been set down as simply imbecile or demented, but a far greater degree of imbecility is seen without any approach to his want of comprehension of words or to the inarticulate character of his speech, and it is not difficult to conceive the helplessness and bewilderment of a man who suddenly finds himself unable to understand what is said, and sees that those around him fail to understand him, while he supposes himself to be speaking as well as ever. Had the patient been an Asiatic, and his only language one of which we were absolutely ignorant, we should not have discovered that he was suffering from any impairment of the mental faculties.

It seems probable, then, that the complete loss of comprehension of words exhibited by this patient would account for his mental condition, and the interpretation here placed upon this loss is that it was due to destructive lesion of an area of the cerebral cortex, in which perceptions derived through the different senses are combined and elaborated into ideas of the objective causes of sensation, which ideas, again, are in this centre associated with and symbolized by names or words. If the interpretation is correct, we have at some parts of the cortex involved in the softening the centre in which "concepts" are formed, "naming" takes place.

It may be considered fortunate that the problem was not complicated by extension of the softening to the third frontal convolution. Had this been the case it would have been impossible to exclude the hypothesis that the amnesia was represented structurally by lesion involving the same cortical area as aphasia but more extensive, and that thus amnesia was an aggravation of aphasia. There being, however, no lesion to which aphasia could be attributed, the distinction, arrived at by analysis, between words considered as intellectual symbols, the elaborated product of sensory impressions, and words considered as motor processes, is confirmed by disease. Dr. Hughlings Jackson has aptly called the third left frontal gyrus the "way out" for words; in the case related it was the "way in," and the centre in which words are formed as "concepts," which were damaged or destroyed.

It is important to note the fact that an affection of language other than aphasia is produced by disease in the *left* hemisphere. The softening was extensive, including the auditory perceptive centre of Ferrier or a part of it (the posterior half of the inframarginal gyrus), the visual perceptive centre (angular gyrus), as well as the more hypothetical centre for concepts and names not yet localized; but affecting one hemisphere only the observed results go beyond the anticipations we should have formed. Still more remarkable is the case mentioned in which a limited lesion in the white substance underlying the angular gyrus of the left hemisphere, and presumably isolating it from this centre for concepts and names, abolished the power of naming an object at sight.

With regard to these, as in aphasia, there arises the question—What would be the effect of a similar lesion in the opposite hemisphere? Destruction of the third frontal convolution on the left side of the brain gives rise to aphasia; on the right side it does not. The precise results of lesion of the right third frontal gyrus, if not simply negative, are not definitely known. Apparently the employment of this convolution in the left hemisphere as the "way out" for language involves the predominant, if not exclusive, employment of this hemisphere at other stages in the complex process by which language becomes the vehicle or, it might almost be said, the instrument of thought.

This, again, according to the hypothesis of Broca and Moxon, is a secondary consequence of right handedness, the dextral pre-eminence of the hand implying sinistral pre-eminence of the brain. It may already be taken as established that there is a marked functional difference between the two hemispheres, which in structure and arrangement are so nearly alike, but whether this consists simply in a relative superiority of the left, or whether there is

thrown upon the right some compensatory superiority in a less conspicuous function than that of language, cannot yet, in the opinion of the writer, be determined. A comparison of the results of lesions in similar parts of the right and left hemispheres will afford information of extreme interest and value. To this Mr. Callender has made an important contribution in the *St. Bartholomew's Hospital Reports*, Vols. III. and V., 1867 and 1869. The question is, however, too large to be opened in the present communication.

A CASE OF PECULIAR AFFECTION OF SPEECH, WITH COMMENTARY

Brain, 1878–9

THIS paper was so far written before Dr. Hughlings Jackson's article "On Affections of Speech from Brain Disease" came into my hands, that to take advantage of it and discuss points in which we appear to differ would necessitate its entire reconstruction. It is, moreover, simply a *mémoire pour servir*, and does not pretend to give a complete account of the relations of derangements of speech with lesions in the brain, but presents the question from a particular point of view. This, it is hoped, will explain and excuse the absence of reference to the work of Jackson, Bastian, Ogle, and others, in the same field, the value of which to science, and its instructiveness to myself, I shall always be forward to acknowledge. The patient, a well-educated and intelligent young man, was admitted under the care of Dr. Sieveking, January 18, 1878.

Eight years previously he had contracted syphilis, and suffered from secondary manifestations of this disease.

For a year he had noticed that wine had an unusual effect upon him, a single glass making him feel intoxicated, and he had had occasional seizures, during which he lost his sight and almost consciousness; his limbs trembled, and he had to grasp something to prevent himself from falling. These fits of *petit mal* lasted from two to five minutes.

About a fortnight before his admission he was suddenly seized with right hemiplegia and loss of speech. It was stated by the friend who brought him that this had come on during the night; but he subsequently explained in a fragmentary manner, that the attack occurred during the evening, that, quarter of an hour before he had been quite well and was smoking; he put down his cigar, and suddenly lost the use of the right limbs.

On admission he could walk in a tottering way, could move the right arm vaguely, hold a pen in his hand, but could not write. He appeared to understand all that was said to him, but was unable to answer questions—he tried, but failed; said "Yes, yes, yes," and would wave his hand. He tried to write with the left hand,

but could not; he produced a letter he had written before the attack to show that he had been able to write.

There was no deviation of the tongue when it was protruded, and he could whistle well, the mouth, however, going slightly to the left.

He improved in all respects, recovering power in the right arm and hand, walking better, and acquiring words. On January 29 he could say his own name, and write it with effort. When asked how he was, he would answer "Yes, yes, very much better," but was capable of little beyond this. Said "yes" on all occasions; in trying unsuccessfully to answer a question would say "Yes—the—Eh! Yes! Yes! Oh gracious me! Yes."

Still unable to read or write.

Dr. Sieveking called my attention to the case on February 26, as one presenting peculiarities, and kindly gave me permission to study and make use of it. By this time the arm and hand had nearly recovered their normal mobility and power, but the leg was carried stiffly, causing the walk to be slow and clumsy. There was no obvious deviation of the tongue on protrusion, or inequality of the two sides of the face.

He remained under observation in the hospital till near the end of March, and was last seen on April 2. The following particulars are summarized from notes made during this period, gradual improvement taking place, but not to such a degree as to cause any essential change in his condition.

He appeared to understand all that was said.

He replied to questions requiring brief and simple answers readily and distinctly, saying, for instance, "Very much better," when asked how he was, and varying the phrase; or, "Oh yes, perfectly," when asked if he understood what he was reading. He would also, when embarrassed in endeavouring to express himself, interpolate the phrase, "but I can speak very much better than I could."

He was unable, however, to give a connected account of anything requiring more than a few words. His method of correcting the statement which had been made to us that he woke up from sleep paralysed and speechless, was somewhat as follows: "No—evening, evening—put down my cigar—smoking, smoking not quarter of an hour—all at once"—indicating by gestures the loss of power in the limbs, and adding—"Couldn't speak."

Again, wishing to inform me that he had heard from a brother in America, to whom a friend had written respecting his attack, he came up to me, fumbling in his pocket for a letter (not the one from his brother, as he told me on inquiry), and saying "Brother—

brother—'Merica—letter—New York—two brothers in America. It was only by questions based on hints afforded by such fragment of phrases that his story was elicited. One of the brothers was dentist in New York. He was unable to recall the word dentis but said "Doctor—Doctor;" indicating, however, that he was nc exactly the same kind of doctor as myself, and getting out of hi difficulty at length by tapping his teeth. When I said the wor "dentist" he assented, and repeated it after me. Another brothe was apparently in Pernambuco; he corroborated my guess tha this was the place he was endeavouring to name, but was neve able to say this longish word.

At first he had lost all notion of numbers, and could not te how many 2 and 2 made; but by practice he could on February add together two low figures, and was beginning to learn th multiplication table. On the 26th he could multiply by 2 and up to 12, but he could not tell what 5×5 gave. He could sho the numbers up to 10 on his fingers, but not 25.

When the table, a glass, an inkstand, violets were pointed ou to him, and he was asked to name them, he was unable to do s When last seen, April 2, he could not name his gloves, or hat, a pen. He named some objects—his hand, the fire, etc.; and whe he had been tested with regard to any particular thing, he woul usually be prepared with its name on a subsequent visit. H was rarely able to give Dr. Sieveking's name, when asked wh physician he was under.

He said he spoke French, and, when tested, did so with an exce lent accent; but in trying to say that he had lived in Paris as boy and gone all over France, he betook himself to English.

The Peculiar Affection.—Up to this point there is nothing ve remarkable in the case. The peculiar and interesting feature w developed when he was made to read aloud. He perused h newspaper regularly, and with all the marks of intelligent interes He understood it also, for he went to the sister in a state of gre excitement to tell her of the failure of a firm with which he ha business relations, carrying the paper in his hand, and pointing o the announcement; and he could always find a given paragraph wh asked to do so as a test. When, however, he was asked to re aloud, the result was gibberish. The following passage was selecte "You may receive a report from other sources of a supposed atta on a British Consul-General. The affair, however, is utterly u worthy of consideration. No outrage was ever intended, and th report is due to misrepresentation of the facts. The Odessa line again working properly." It was read slowly, and in a jerky mann as nearly as it could be taken down thus: "So sur wisjee c

wenement ap ripsy fro fruz fenement wiz ā secona coz foz no Sophias ā thee freckled pothy conollied. This affaise eh oh cont oh curly of consequences. Uce sudos val oh es es entain ah hee enepol ā oh dee ā ah messequence oh coz foz. The assoil lens ā puff pifl miss corres povety."

It will be seen that there is no traceable relation between the passage and his rendering of it beyond a certain imperfect correspondence between the number and length of the words, obscured by the difficulty of determining whether some of the sounds represented words or interjections thrown in when he was perplexed. Unfortunately he was not made to read the same passage twice or three times, his attempts being taken down in writing, but it was clear that there was no regularity in the substitution of certain constant inappropriate sounds for given combinations of letters.

It was evidently an effort to read aloud, requiring close attention, and he read seriously and steadily, apparently unconscious of the absurdity of his utterances (probably because he was too intent on his task), till interrupted by laughter which it was impossible to restrain, in which he usually joined.

He was never able to give the simplest written answer to a question or to write from dictation, but he signed his name quite well, and wrote down the names of his brothers, but with the initial only of the Christian name, the surname in full. He wrote T. for Tom, but could not write Tom, though habitually saying it. He could not give his address in writing.

When asked to copy a sentence, he wrote the short words quickly and in a good hand, but a long word he took down slowly, letter by letter, in large schoolboy characters, usually accurately; but as he wrote each letter he named it aloud, and *always wrongly*. He always, however, gave it the name of some letter or other, and not a name of his own invention.

Commentary.—At first sight it seems impossible to realize the mental condition in a case such as this, or to specify the derangement in the mechanism of speech and thought which gave rise to the extraordinary perversion in the translation of printed words into sounds; but cases have been observed which appear to throw, at any rate, some light upon the phenomena.

It will be well, in the first instance, to estimate as nearly as possible the general intellectual damage. This appeared to be very slight. The patient understood all that was said to him, conducted himself rationally, appeared to have perfect recollection of his past life and occupation, displayed an intelligent interest in all that was going on around him, in his own case and in our inquiries into it, and showed much ingenuity in contending against the diffi-

culty in expressing himself occasioned by his loss of memory for words. He appeared, however, to have lost the idea of number to a very considerable extent; he could not at first have added 6 and 5, or told what twice 6 made; and when he had mastered the first two lines of the multiplication table, he was still unable to say what 5 times 5 gave. Possibly this might arise from failure to realize the number indicated by its name, or to recall the name of a number, and be an example of his loss of this memory for names. Unfortunately he was not tested by concrete illustrations, as by being made to give change for or out of a sovereign. He was unable, however, as already stated to show on his fingers numbers above 10.

The difficulties in expressing himself in words seem to be capable of resolution into the following factors.

1. *Ideas no longer evoked in the mind the words necessary for their expression.*—So far as this went, it was a loss of memory for words, and not the loss of the memory how to say words; if the words were supplied, or merely suggested, the patient made use of them at once. In this respect, therefore, the case was distinguishable from one of aphasia proper, in which phrases or words dictated cannot be repeated. It was not, however, simply a loss of the memory of names or of the power of naming. In a spoken sentence there are two distinct factors, the names, and the other parts of speech which bring the names into some relation with each other (a proposition consists of a subject and a predicate); the names alone are intellectual symbols of external objects or of concepts; the predication in which the other words are employed is an intellectual operation. Now in some instances of loss of memory of words the difficulty arises entirely from the impossibility of recalling to mind names (a condition which might be called simple amnesia had not this term through vague employment lost all definite meaning). Such was not quite the case here; the patient had difficulty in remembering names, but often when he appeared to have the names in his mind, as for example, "brother," "New York," "America," he could not construct the sentence.

2. There was therefore also a defect of "intellectual expression," that is, in the efferent department of the cerebral mechanism of speech, but anterior to that which constitutes simple aphasia. An aphasic may propositionize in consciousness, may mentally rehearse a sentence but be unable to utter it; the patient here was obviously unable to construct a sentence in his mind, but once present to his mind it was promptly spoken.

3. *The sight of an object no longer recalled its name.*—This might be looked upon as simply a particular case of the general defect of memory for names which existed here. But the faculty of naming

objects at sight (the automatic suggestion of the name by a visual impression) may be absolutely lost, while there is little or no impairment of the power of recollecting names or other words.[1] A case is reported by me in Vol. LV. (1872) of the *Medico-Chirurgical Transactions*, in which a man who talked fluently and well, and was scarcely ever at a loss for a word of any kind, could not name the most familiar object shown to him. He could not give the name of a single letter and could not therefore read; he could, however, write correctly, either from dictation or spontaneously, but he was absolutely unable to read what he had himself written.

4. There was absolute loss of the faculty of translating auditory symbols (spoken words) into visual or graphic symbols, and of the power of graphic expression, i.e. he was completely agraphic; could neither write from dictation nor spontaneously, though he could copy and render printed into current characters.

5. While he understood visual or graphic symbols he could not render them by the appropriate verbal symbols.

It is no explanation of cases such as this to invent names more or less expressive of the derangements observed or to describe them in psychological terminology. What is wanted is that the damage in the cell and fibre mechanism of speech, and of the mental operations concerned in speech shall be specified. This mechanism is, however, yet unknown: a few important portions of it are identified, but that is all which can be said.

The Mechanism of Speech.—In attempting to construct this mechanism hypothetically, the basis must be such facts of structure as are known, together with experimental and clinical localization of functions; and the scheme must be consistent with the mode of nervous structure and operations as exhibited in the simple forms of nervous apparatus.

We have throughout the nervous system a receptive and emissive department, which may be called, for convenience, sensory and motor respectively. The relations between these two divisions, and between higher and lower nerve-centres, as exhibited where they are simplest and most easily followed, viz., in the spinal cord, will furnish a guide to the higher and more complex relations.

Now all muscular movements are performed under the direction of a "guiding sensation." The precision of action of the limbs would be impossible without the controlling influence of sensation in the parts set in motion. Structurally this would be represented by saying that the motor cells of the anterior nerve-roots concerned

[1] The converse, however, of this—absolute loss of the power of calling to mind names while the name is at once suggested by the sight of the object—is not known to me.

in the execution of a given movement are originally selected and combined through the sensory cells of the posterior nerve-roots. For each movement there is employed a certain definite group of motor nerve-cells in the cord; the education of the cord for a given movement would be the association by processes of the cells forming its group; and this is effected under the influence of guiding sensations from the limb, i.e. through the sensory cells in the posterior grey columns. The motor cell-group constituted in this way is then ready for employment by a higher centre.

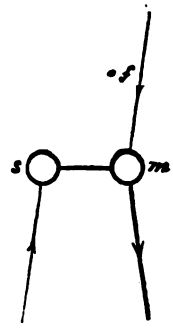

FIG. 8.

A diagram may assist in rendering this idea more clear. *s* is a group of sensory cells, which receives the guiding impressions from the periphery, and under its influence is arranged the group of motor cells *m*, which call into action the requisite muscles; *f* is a bundle of fibres, from a higher centre which employs the motor group *m*. This is representative of the entire process of motor education. A motor cell-group is formed under the guidance of a sensory cell-group on the same level, and, when formed, is made use of by a higher centre; this higher centre having, so to speak, been the exciting cause of the process. An infant in the dawn of its intelligence desires to use its hands to grasp an object and carry it to its mouth, this is the stimulus from the higher centre; the gradual acquisition of precision in the required movements is the association of the appropriate motor-cells in the cord for combined action, which is effected through guiding sensations from the hands and arms, transmitted of course to the sensory cells, and by these to the motor-cells. (We may parry the objection that sight is the guiding sensation, by supposing the child employed in the illustration to be blind.)

In the case of words, there will be somewhere or other in the

higher centres a set of motor nerve-cells, from which will descend fibres to the nerve-nuclei of the thoracic muscles for the production of an expiratory current of air; others to the larynx for phonation, others, again, to the tongue and lips for articulation. The set of cells called into action in the utterance of a given word will constitute what I have elsewhere called a "word-group," and I have hypothetically located such word-groups in the corpus striatum, where they would be formed or linked together under the guidance of auditory sensory impressions, reaching the corpus striatum through the thalamus. The word-groups in this ganglion would be simply motor combinations, quite irrespective of the use of words in language, intellectual and emotional; they would, in fact, be exactly on the same level as the words learnt by a parrot.

I am no longer, however, quite so confident that the corpus striatum is the indispensable intermediary between the hemisphere and the cord, since cases are on record in which both divisions have been destroyed or greatly damaged without the hemiplegia which would be inevitable were this the case. This, however, would only shift the seat of the primary word-groups to the third left frontal convolution, without making any other difference in the plan of the mechanism of speech, which I advance by way of provisional hypothesis.

Ascending to the hemispheres, it will be simply assumed without argument that the third left frontal convolution is the "way out" for intellectual expression by speech, for words in relation to ideas. This is not simply because the third frontal convolution is the convolutional motor centre for the lips and tongue, since we constantly see aphasia without right facial hemi- or monoplegia and conversely right facial hemi- or monoplegia, without aphasia. The higher centre which calls into action Broca's convolution we cannot localize. The guiding centre will be the auditory perceptive centre placed by Ferrier in the inframarginal gyrus of the fissure of Sylvius. The cells of the third frontal will be trained and grouped for the utterance of ideas transmitted to it for expression, by means of cells of the Sylvian inframarginal, which, as the seat of auditory perception, will have registered words as heard.

Affections of Speech.—Now just as the integrity of the nervous apparatus of guiding-sensation is essential to precise and orderly movement, even when the motor cell-groups are organized, so is the due connexion between the auditory perceptive centre and the speech-centre essential to precise and orderly utterance. Damage to this relation is the explanation of "mistakes in words." For the sake of clearness we may again resort to a diagram.

Lesion of *s*, the speech-centre, the third left frontal, will give rise to aphasia proper; lesion of A, the auditory perceptive centre (inframarginal Sylvian gyrus), or of *a s*, A and S remaining intact, would cause "mistakes in words," probably different in kind in the two cases.

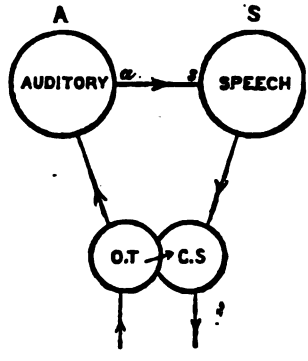

FIG. 9.

Up to this point we have the explanation (hypothetical and provisional) of *three forms of affection of speech.*

1. In the lesion of S, of simple aphasia, in which the patient understands all that is said to him, and knows what he would like to say in return, but cannot put it into words; the "way out" for words in intellectual expression is broken up. He may say "Yes" and "No," and a conventional word such as "Good-bye" may slip out when occasion arises, or an emotional phrase on excitement, which I have elsewhere supposed to be possible through the existence of primary word-cell-groups in the corpus striatum, which had, by frequent repetition, acquired a secondary reflex association with emotional states. He will be unable to repeat his favourite oath, or his formula of welcome or leave-taking, or perhaps even "yes" and "no," when bidden to do so.

2. By lesion of the line of communication as the explanation of mistakes in words recognized by the patient.

3. In lesion of A of mistakes of which the speaker remains altogether unconscious.

There are higher centres, however, than those with which we have been occupied, and we may consider the part they play in the function of language, and the disorders which lesion of them may produce, before we refer to the complications introduced by reading on the one hand and writing on the other.

Naming and Propositionizing Centres.—The formation of an idea of any external object is the combination of the evidence respecting it received through all the senses; for the employment of this idea in intellectual operations it must be associated with and symbolized by a name. The structural arrangement corresponding to this process I have supposed to consist in the convergence from all the "perceptive centres" of tracts of fibres to a convolutional area (not identified), which may be called the "Idea Centre" or "Naming Centre." This will be on the sensory, afferent, or upward side of the nervous system; its correlative motor centre will be the "Propositionizing Centre," in which names or nouns are set in a framework of other words for outward expression, and in which a proposition is realized in con-

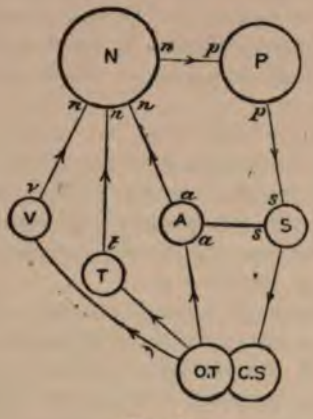

FIG. 10.

sciousness or mentally rehearsed. If we are to have a seat of the faculty of language, it would be here rather than in the third left frontal convolution, with which, however, it may possibly be in close proximity.

Expressing this by a diagram, we have V, A, and T, the Visual (angular gyrus, Ferrier), Auditory (inframarginal Sylvian gyrus), and Tactual (uncinate gyrus), perceptive centres sending converging tracts of fibres, *v n*, *a n*, *t n* to N, the "naming centre." Here the perceptions from V and T (smell and taste are omitted for the sake of simplicity) are combined into an idea, which idea is symbolized by the word reaching N through A, which has always, in the experience of the individual, been associated with the object.

P is the propositionizing centre in which the phrase is formed, it relations with N and S being sufficiently clear.

It will no doubt be objected that there is no evidence of two centres, one for propositionizing, and the other employed by it for utterance; one for the construction and mental rehearsal of a phrase, and the other for realizing it in spoken words; and it is unquestionably true that, in a large proportion of cases of aphasia both faculties are lost. The independence of the two operations seems to me, however, to be established by the following facts: *a*. There are cases in which the patient cannot frame the simplest phrase, but can repeat anything said before him. Here A is intact, N destroyed. *β*. The converse of this—namely, that a proposition is realized in consciousness, so that not only the wish, but the words, are present to the mind, while the expression of them is impossible—is difficult to establish; but a comparison of cases of aphasia has led me to the conclusion that such is sometimes the case. If, however, there are really instances in which speech is lost while the power of writing is retained, this would be conclusive. This will be considered later.

This diagram will perhaps bring out clearly, what is, however, well recognized, the distinction between nouns or names and other parts of speech; the receptive centre, N, has to do only with names; as soon as other words are brought into use a motor function is exercised. It will further enable us to represent the causation of *other derangements of speech*, e.g.—

4. Loss of the memory of names or nouns. Numerous examples are on record; and a very good illustration has lately passed under my observation. An old gentleman, after very slight right hemiplegia, can give long answers fluently and volunteer statements, so long as the phrase does not contain a noun. "Oh yes, I am much better than when you last saw me." "I shall be 73 on the three—four"—when he confused himself in trying to find the word December. He could not name a hand when told to do so, but in his effort something like leg was once heard. This gentleman's memory of facts, events, dates, and faces is very good.

It will be at once seen that lesion of the naming centre, N, gives the explanation of the functional loss.

5. Another derangement of speech is where names are more or less remembered, but there is loss of the faculty of constructing a sentence which shall convey the ideas to be expressed regarding them; that is, of the power of framing a proposition. The lesion here will be in the propositionizing centre P.

This condition is illustrated imperfectly in the case which forms the text of this commentary, as when the patient could only say,

"Brother, brother—New York—America—two brothers in America—letter," leaving it to us to fit the words together so as to convey information.

6. Still another derangement of language is one well illustrated by the case to which allusion has already been made, related in my paper "On the Mechanism of Speech and Thought," in the *Medico-Chirurgical Trans.* for 1872 (Vol. LV.), and dwelt upon also in a communication "On a Case of Amnesia," in the *Transactions* for 1877–8, that of a man who conversed fluently and wrote correctly, both from dictation and spontaneously, but who was unable to name any object which he saw, or to read a single letter, printed or current, even in what he had himself just written.

The channel of communication between the visual centre, V, and the naming centre, N, was destroyed. It may be added that the lesion found in this case underlay the angular gyrus in which Ferrier locates the visual perceptive centre.

Traces of a similar loss of the power of naming an object at sight are present in many recorded cases, but it is rarely that the clue has been followed. In the case here related there was impairment of this naming power.

7. Cases in which the auditory perceptive centre A, or its communicating tract with the naming centre *a n*, is destroyed. These have been recently considered in the communication to the Medico-Chirurgical Society, already mentioned "On a Case of Amnesia, etc."

If the speculations here pursued are well-founded, and it is claimed for them that they are based upon careful anatomical investigation, and upon the application to the results of this investigation of the localization of functions by Ferrier, Hitzig, and others, to which the anatomical facts adapt themselves with perfect readiness, it follows that derangement of speech may be caused by lesions in different parts of the cerebral cortex, as has been found on post-mortem examination. In the case of amnesia reported to the Medico-Chirurgical Society, in which there was loss of comprehension of spoken or written language, while the speech was mere gibberish, the lesion lay entirely behind the motor region of the hemisphere as had been prognosticated.

Now most of the objections to the functional association of Broca's convolution, with an important link in the mechanism of speech, are based upon the idea that all forms of speech disturbance should be accounted for by changes in this gyrus. This idea has been encouraged by the vague use of the term aphasia, and by employing compounds of this word, such as amnesic aphasia, ataxic aphasia, paraphasia, etc., to denominate

different speech-derangements. It cannot be too strongly urge that different affections of speech indicate damage to differen parts of the mechanism of speech, and therefore lesions at differen points of the cortex, or white fibres of the hemispheres. In cours of time, by careful analysis of derangements of language and speech and by minute and accurate description of the lesions associate with definite forms of derangement, the localization of the differen cell-areas which enter into the apparatus of speech may be effected

Conjectured site of the Propositionizing and Naming Centres.—It is probable that the propositionizing centre P, and the speec or utterance centres, are in very close proximity, as they are so com monly affected together; and P, as belonging to the motor departmen of the cerebrum, would be in the anterior part of the hemisphere N, on the other hand, being on the reception or sensory side of th nervous apparatus, might be expected to be in the neighbourhoo of the perceptive centres. As a provisional guess I have conjecture that S may be in the foot of the ascending frontal (first ascendin parietal) gyrus, P in the third left frontal, and that N may be situate in an unnamed lobule (collateral lobule of my paper "On th Structure of the Cerebral Hemisphere"), situate on the under-surfac of the tempero-sphenoidal lobe, near its junction with the occipit lobe, as fibres from all the convolutions in which perceptive centr have been placed by Ferrier converge to and end in its grey cortex.

Reading and Writing.—There remain to be considered the con plications introduced into the question of language by readin and writing. They are extremely perplexing, and have bee arbitrarily excluded from consideration up to the present stage the discussion, for the sake of simplicity and clearness.

The steps of the educational process by which these arts a acquired are as follows: Reading of course precedes writing, an there will be first the association of visual perceptions, letters words, with previously familiar sounds, or auditory perception the acquisition of this association is greatly facilitated, and almost aided by simultaneous utterance of the sounds at the biddi of the teacher; but this is not absolutely necessary, as childre sometimes learn to point out letters which they cannot be persuad to name. This process will be represented in the diagram by brin ing into use fibres connecting the auditory and visual percepti centres A and V, and again perhaps by fibres passing between t visual centre V and S, the speech centre.

At first the entire attention is absorbed by organization of t association-links between these lower centres, and the subjec matter of what is read is altogether neglected. Later, when t association of the visual and auditory perception of words

perfected, and the response of the speech-centre S has become automatic, written words convey ideas to the higher centre N, and eventually quite as promptly as spoken words. In reading to oneself, the path V S may come to be as entirely neglected as the path A S in listening. Whether the perceptions are transmitted upwards through A, or directly from V to N, cannot be said. Since, however, it appears from observation of cases of aphasia that visual perceptions of words, printed language, must be realized, or capable of realization, in consciousness as spoken words in order to be understood, it does not seem probable that an independent path exists between V and N.

In learning to write, the centre W, which will be that for the delicate movements of the hand, is educated through the visual centre V; that is, by repeated copying, cell-groups, or cell-groupings, will be organized in W, representing written words which can then be employed by the propositionizing centre P in intellectual expression. There must, however, be a close association between S and W.

As a rule, aphasia and agraphia go together, and this all but constant association is not explained by any very close proximity of the centres for the lips and tongue to that of the hand, as localized by Ferrier and Hitzig, causing the two to be involved in a common lesion. There may be absolute loss of the power of writing when the lesion is limited to the foot of the ascending frontal and the posterior part of the inferior frontal gyri, quite away from the part of the ascending convolutions in which the hand centre is situated.

Either, then, the fact that writing is a form of intellectual expression engrafted upon speech makes one centre, W, dependent upon S, or speech, S, and writing, W, are both employed by some higher centre, P, in which ideas are primarily clothed in words. Now writing is sometimes lost when speech is not, or more profoundly affected, as in the case related, and in many others. This would fit in with either hypothesis. If, however, there are cases in which a man can express himself in writing but not in spoken words, the lesion not being in the pons or bulb but in the hemisphere, the second supposition must be the true one. Such a case I have never seen, though I have had a patient in St. Mary's Hospital who could write better than he could speak, whether in answering questions or on dictation—and very few of the examples on record will stand a critical examination. There is no post-mortem evidence on the subject known to me.

The case of Dr. Osborne's, quoted by Dr. Bastian,[1] seems at first sight to be an instance; and as it has points of resem-

[1] "On Various Forms of Loss of Speech." *Medico-Chirurgical Review* for 1869.

blance with the case here related, a brief summary of it may be given. The patient, a well-educated man, understood all he heard and read; had lost none of his mental power; could construe Latin and Greek, and work out mathematical problems; and, finally, *could write fluently*; his speech, however, was absolutely unintelligible; and when he was made to read aloud, it was pure gibberish, except that such words as "in the," "to," and "as," were often correctly given. Here the lesion was obviously not bulbar but in the hemisphere, and there is clear evidence of derangement of articulate expression; while there was no corresponding impairment of the power of expressing ideas in writing, which would seem to furnish the proof required of independent centres for speech and writing. My explanation, however, of the phenomena is that the speech centre was cut off from its guiding influence, the auditory perceptive centre, and not that the speech centre itself was damaged, i.e. the tract A S was destroyed.

It has been necessary to explain the provisional hypothesis of the mechanism of speech which has grown up in my mind, but it is not attempted to do more than give a sketch of it and we may now apply it to the case under consideration.

Analysis of the case.—In the first place the speech centres was undamaged. When words occurred to the patient's mind, or were spoken before him, he could utter them without difficulty. There would be no lesion of the third left frontal gyrus.

The propositionizing centre, however, was affected: he was unable to frame any but the most simple phrases, even when the nouns were supplied, and he knew well what information he wished to convey.

Writing was entirely lost. He could copy, though with difficulty. Could sign his name and write down the names of his brothers, but with the initials only of the Christian names. He could not answer the simplest question in writing, even when "yes" or "no" only was required. Could not write a single word from dictation, and could not even write "Tom," which he told me was the name of one of his brothers, though he wrote the initial.

It is easy to see that the agraphia depended upon failure to realize mentally written words, or to call up a pictorial representation of them, which is a necessary antecedent to their reproduction in writing. This was evident from what was observed when he was made to copy; such words as "it," "the," "and" etc., which he could carry in the mind for the requisite time, he wrote rapidly and in a good hand, while longer words were taken down slowly, letter by letter (each one misnamed aloud) and in awkward schoolboy characters. As mental pictorial representations will be repro-

duced where they were originally engraved by perception of the words as external objects, we trace the derangement to the visual perceptive centre, which will be seen to be the starting-point of other derangements.

We must stop for a moment, however, to remark upon the contrasts between this case and one of those so frequently mentioned in this communication, that in which a man had lost, absolutely and entirely, the power of naming an object at sight and of reading, while he could not only talk fluently but write quickly and well. The present patient, who had also lost in a great measure the power of giving names to objects seen, could, however, understand what he read, but, on the other hand, could not write. These differences, perplexing at first sight, are easily explained. In the first case the visual centre was uninjured and was in communication with the writing centre, while it was cut off from the naming centre (it may again be mentioned that on post-mortem examination of this case the angular gyrus was healthy, but the white substance of the centrum ovale immediately subjacent was softened); in the present case the visual perceptive centre itself is damaged and all operations in which it takes part are impaired.

There is a further interest in the analysis of this agraphia, as it furnishes a kind of parallel to some cases of aphasia; the patient could frame a simple proposition in words, but could not express it in writing. We have in this proof that words as *intellectual symbols*, i.e. as vehicles of ideas, may be present to the mind while it is impossible to call up words as *visual symbols*, as groups of printed or written characters (the term "symbols" is used here advisedly, in order to bring out the double sense in which it is employed). In the same way we may have present to the mind ideas and words in which they could be expressed, but not words as sounds.

Returning to the analysis of the phenomena presented by this case, it is evident that the naming centre was affected, and that the difficulty of recalling nouns was not simply the fault of the visual centre. The patient was frequently at a loss for words (nouns), and had to resort to pantomime or circuitous expressions; and it will be remembered that there may be perfect readiness in conversation when the name of the most familiar object is not recalled by the sight of it.

There remains to be considered the extraordinary perversion of speech which resulted when the patient was made to read aloud. This was clearly not due to any affection of the speech centre itself, or to derangement of the downward communication to it from the propositionizing centre. In reading aloud there is a double opera-

tion, the apprehension of the meaning of what is read—in other word the transmission upwards of the visual perceptions of words to tl centre in which they are translated into ideas—and the translatic of printed into spoken language, i.e. the revival in consciousness the auditory symbols corresponding to the visual symbols, and tl utterance of the former. Clearly the bungle took place in tl realization of words as auditory symbols, when presented as printe characters, which ought to be an automatic operation, and the inju done must have been to the association between the visual ar auditory perceptive centres, probably from mischief in the visu centre itself, since there were other evidences of its function derangement.

I should localize the lesion in this case with some degree of co fidence. The history shows the disease to have been syphiliti and its result was no doubt closure of the middle cerebral arter The mode of access seems to indicate that at first the main vess was occluded; but the degree of recovery shows that circulati was re-established in the branches to the corpus striatum, tl third frontal, and the anterior extremity of the temporo-sphenoid lobe, which are given off in the first part of its course. The pers tent impairment of function was chiefly in the area of the branch which issue from the posterior part of the fissure of Sylvius to supp the supramarginal and postero-parietal lobules, the angular gyr the posterior part of the inframarginal and parallel gyri and t collateral lobule of the temporo-sphenoidal lobe. Whether t artery was blocked before these branches arose from it, or each o of them was separately involved in a small gummatous growth it emerged from the Sylvian fissure (as I have sometimes see cannot be stated definitely. The lesion does not appear to ha amounted to complete softening.

REMARKS ON SOME AFFECTIONS OF SPEECH

British Medical Journal, 1907, Vol. I

Dr. Thomas, Assistant Medical Officer of the London County Council, relates a number of interesting examples of speech defects met with in children.

In one series it is chiefly in reading that the difficulty occurs. An intelligent boy of 7 years old cannot remember how to make l, p, y, g, that is, cannot reproduce the written or printed characters representing the articulate sounds, and reads, "It has three birds in it" as "To see best in to," that is, he cannot reproduce the articulate sounds represented by the printed words. On the one hand, he cannot translate auditory impressions or perceptions into visual representative characters, and, on the other, cannot translate visual symbols into the appropriate articulate sounds. He recognized words if they were spelt to him; he did difficult sums in mental arithmetic, and when 11,308 was written in numerals gave the number in words.

Another boy could not read the smallest words. Said "chair" spelt "sister"; made serious mistakes in writing from dictation; did not copy correctly. He, also, calculated correctly, and was said to "know all his letters," that is, I suppose, could name them, or perhaps could say the alphabet.

A third boy, of 11, good at arithmetic, smart, ready, and trustworthy spells out "girl" as "vice"; cannot read "mouse," though he recognizes each letter; writes from dictation "a pen and ink" as "a gen and inck." It may be accidental that the two words which he bungles are nouns, while "and" which has the same number of letters, he writes correctly.

These, with a case which has come under my notice, afford an opportunity of discussing the terms "*word-blindness*" and "*letter-blindness*," and appear to me to be worthy of consideration as bearing upon the mechanism of speech and thought.

My case is as follows —

I was informed that a boy of 14, of superior intelligence, was kept in a lower class at school because he could not read aloud,

and that his prospects in life were likely to be injured by his not reaching the proper standard.

I found him to be remarkably alert intellectually, fond of reading, particularly adventures and travels, full of information, ready and apt in conversation, and altogether a very interesting boy. He could not explain his difficulty in reading aloud. I gave him two long and rather complicated paragraphs from the *Times* to read, and asked him to tell me what they were about. This he did readily and clearly. I then asked him to read them aloud. His face at once took on an expression of anxious effort. He did not read even short words fluently, but uttered two or three with the slight hesitancy and uncertainty of a beginner, and was then brought to a standstill for a moment by some longer word, not because it was difficult or unfamiliar, but apparently as if he had to grasp its meaning and pass it through his mind for utterance. Sometimes it seemed as if he were on the point of substituting another word (Dr. Thomas's boy No. 1, when shown the word "buds," read it "branch," which looks like an intellectual substitution).

There was here clearly no word-blindness in the true sense of the term, since that is a purely sensory defect, and this was a motor failure, not a sensory defect. Words printed or written reached his mind with normal readiness, and were perfectly understood. There was no fault in the mechanism of speech or intellectual expression, to employ the excellent term introduced by Hughlings Jackson. The afferent and efferent paths, and the sensory and motor centres concerned in language as an intellectual process were intact. It was utterance in one particular respect which was interfered with—the translation of visual characters into the articulate sounds which they represented.

This boy ultimately gained the faculty of reading aloud fairly well, but with great effort, and when he read aloud the attention was so concentrated on the task that he did not understand what he read.

Reading aloud.—Now this is not an intellectual operation properly speaking. We can read aloud quite easily an unknown language—it is a mere matter of putting into corresponding spoken words certain combinations of letters. No ideas reach the mind. So, again, we can read aloud, often with appropriate inflection of the voice and emphasis, while thinking something else all the time, and not at all realizing mentally the meaning of what we are reading. The same convolutions are not engaged in translating printed words into articulate sounds as are concerned in the intellectual recognition of the meaning of the word and its employment in the mental operation which ultimately finds expression in speech.

A case closely parallel to this came under my observation in 1878, and was related in *Brain*, vol. i. The patient read his newspaper with great interest, and related the substance of paragraphs to the sister of the ward and to the class, but when asked to read aloud, the result was a jargon which had no recognizable relation whatever to the words he was reading.

The printed words reached his mind and conveyed their meaning, but the cell and fibre mechanism by which the association between visual word symbols and the articulatory motor centre is effected was damaged.

In another case an old woman in reading aloud a familiar verse, which she understood perfectly well, always read it wrong, but differently every time; there was, however, some correspondence in length and sound between the words as she read them and the words of the verse.

Another case seen in 1871, and related in a communication to the Royal Medical and Chirurgical Society "On the Mechanism of Speech and Thought," corresponded more closely with Dr. Thomas' cases, supposing, that is, that boy No. 1, while still failing to name letters and to read aloud, did not learn the meaning of the words.

The patient, a very intelligent man, could not read either printed or written characters, except his own name. He could write letters to his wife, or from dictation, and took elaborate notes of my instructions, but could not read even what he himself had written.

The Mechanism of Speech and Thought.—The conception which I entertain of the mechanism of speech and thought is based on the functional and structural arrangements of the spinal cord.

Throughout the nervous system there is an afferent or sensory and an efferent or motor side. All movements are in orderly response to sensory impressions; the sensory impression guides and governs the movement which it calls into action. The structural representation of this physiological fact is that sensory cell-groups arrange and combine the motor cell-groups which put into execution the dictates of the sensory impressions. Such motor cell-groups may be said to represent the words. In the cord the sensory and motor cell-groups execute reflex actions which may be extensive and elaborately co-ordinated.

At the next higher level the sensory impressions and movements are re-represented, to employ Hughlings Jackson's useful term, and undergo an intellectual elaboration. Impressions are translated into sensations, and the sensations, tactile and other, derived from the skin, muscles, and viscera, are co-ordinated with visual sensations in such a manner that visual and tactile form and size correspond. The tactual centre may be said to educate the visual

centre. The movements of the limbs and of the body general are at this level brought, in some degree, under the guidance visual sensations.

The spinal motor centres have been educated by the correspondi spinal sensory centres, and large and complex cell-groups have be formed representing extensive co-ordinated purposive movement These groups are placed at the service of the visual sense-cent which can call into action and combine for a given purpose, und the guidance of vision, movements of the entire body and limb It may almost be said that the spinal motor centres conce themselves with the muscles, while the visual centre directs mov ments. The second level in which the primary intellectual elabor tion—the translation of impressions into crude sensations, a the organization of movements under the guidance of vision- takes place is, in my judgment, constituted by the great centr ganglia, the thalamus (sensory) and corpus striatum (motor). is interesting to see that these two great masses of grey substanc which have long been almost ignored by neurologists, are bei reinstated in their important position.

Another step in intellectual elaboration is perception, the reco nition in consciousness of the external object to which the senso impression is due. This is done separately for each sensation, a we have a corresponding number of perceptive centres, tactu visual, auditory, gustatory, olfactory—all of them in margin convolutions which are reached by radiating fibres ascending fro the central ganglia, bulb and cord—each of which directs and co trols motor centres, also situate in marginal convolutions and relation with radiating fibres, which, however, are efferent. T movements of the body and limbs are under the joint control the tactual and visual centres, the tactual perceptive centre bei concerned in what may be called individual movements, the visu with general combined movements.

The auditory perceptive centre while in relation with the mot system generally and automatically for protective purposes, when we start at a noise, or turn aside, or assume certain attitud has a special educational relation with the muscles concerned speech. Words, as articulate sounds, are acquired by imitatio they can, to a certain extent, be so acquired by a parrot or rave Articulatory motor cell-groups are organized in the left third front convolution under the guidance of sensory cell-groups in the au tory perceptive centre, the superior temporal convolution. Wor are thus primarily automatic—percipio-motor processes onl parrot-like reproductions of articulate sounds. Words of an u known tongue listened to and uttered are nothing more. Th

employment as intellectual symbols, as having a meaning, as vehicles and instruments of thought, although taking place as the child is learning to talk, is another matter altogether.

Reading.—In learning to read, the process is at first, while relating only to letters, almost more distinctly concerned with perceptions, as distinguished from ideas, and is more independent of intellectual operations, properly speaking, than that of learning to talk. In learning to speak, a double process is going on, for while the utterance of a name is gradually accomplished by the organization of a communication between auditory and articulatory cell-groups, the person or thing designated is before the child's eyes, and is associated in the child's mind with the name. The name comes to represent a mental image, and is not merely an articulate sound.

The letters that a child painfully learns to associate with the appropriate sound do not raise any idea whatever in the mind. The process is simply the association of an arbitrary visual character with a given auditory perception. There is no natural connexion between the two, but an association is established by an effort of attention. The letter has to be repeated aloud again and again. The association between the printed character and the sound which it represents—between the visual impression and its auditory equivalent—is reinforced by the articulatory rendering of the sound which brings into action the imitative motor process. There is no such thing as a separate centre for letters as such; a letter is learnt when such an association is formed between the visual and auditory centres that the character seen at once suggests the particular articulate sound. This connexion may be established with difficulty and imperfectly, or it may be deranged by some lesion, so that the individual cannot name the letters shown to him. This has been called "letter-blindness"; but it is not a sensory defect at all—it is the result of an imperfect sensori-motor mechanism. A word made up of these very letters may convey an idea to the mind.

The sensory centre for printed or written words is the same as that for individual letters, and the translation of words into the appropriate articulate sounds in reading an unknown language is exactly the same process as learning the individual letters. It is the establishment of an association between visual characters and an already organized articulatory motor centre. It is on a level with parrot-like speech. But before the child is taught to read, spoken words have become intellectual counters. The name of an object calls up a mental image formed by the convergence on a higher cortical centre of all the perceptions to which the object gives rise—a centre in which the name becomes the symbol of the

combined perceptions, or complete image or idea of the object. This, which is sensory in character, and constitutes the cortical summit of the afferent side of the nervous system, has a corresponding cortical motor centre for intellectual expression which it educates and employs, and this in turn makes use of the word groups ready organised by the auditory perceptive centre in the left third frontal convolution, the downward starting point for words, for articulate speech. These two centres I have called the naming centre and the propositionizing centre respectively.

The word groups, then, are called into action by impulses along two different routes, a low level route from the auditory or visual perceptive centre, and a high level convolutional route, which, starting in the auditory or visual marginal perceptive centre, ascends to the centre for names or ideas, and is transmitted forwards to the centre for intellectual expression, which employs the word groups for utterance.

An interesting question here arises. When a printed or written letter or word is read aloud, does the visual perceptive centre reach the motor word group in the third frontal, organized by the auditory centre, by fibres passing directly to them, or does the word as seen call up the word as heard—that is, the corresponding sensory word group in the auditory perceptive centre, and make use of it to pass on the impulse which gives rise to the uttered word to the motor word groups? The latter is the order of events which probably makes the difficulty in reading aloud to be due to imperfect association between the visual and auditory perceptive centres. It is not likely that fibres pass from the calcarine or occipital region to the third frontal, whereas the calcarine and angular convolutions are adjacent to each other. It is true, however, that a deep-seated mass of commissural fibres takes origin in the occipital lobe, and proceeds towards the frontal convolutions.

Sufficient has been said to explain the idea which I entertain of the particular imperfection in the mechanism of speech of which the cases related are examples.

Numerals.—The question of numbers in relation to aphasia of various kinds is one of very great interest. It was very early found that some patients whose vocabulary was limited to "yes" and "no" and a few interjectional exclamations could state distinctly the number of objects given them to count. Sometimes the numeral would drag the name of the object with it, as, for example, a man who could not say "shillings," when asked to repeat it, or when shown the coins, said "five shillings" after counting this number when the amount was placed before him. A patient who could not say any part of the alphabet could count up to twenty, and,

while unable to write a single letter, would make figures on the palm of his hand with his finger to show the number of spokes in a wheel.

These are instances of motor differences between words or letters, and figures. Even more remarkable is the sensory difference between letters and numerals. Dr. Thomas' boy, who could scarcely recognize letters, at once put into words "11,308." From such examples as this it has been inferred that there is a different cortical centre for letters and figures. But, suppose this had been a Roman boy, would he have failed to recognize "X" as signifying "decem"? He would have been unable to say "x," to translate a visual character into an articulate sound, but he would at once have interpreted it as signifying "ten," and said "decem." It is not that there is a separate centre for letters and numerals, but that they represent different cerebral processes.

A number is not like a name or noun; it does not call up the image or idea of any particular object; it has nothing to do with the properties of objects, and it is not a motor part of speech. It represents, in a sense, an abstract conception, but not of a very high order. Dogs can probably count sheep, birds can count the eggs in their nests, and other instances of the faculty of enumeration by animals might be cited. A calculating boy need not be otherwise very clever. The auditory and visual perceptive centres receive and register spoken and written numerals, and the word cell-groups for the utterance of numbers must be organized in the articulatory centres; but addition and subtraction and other arithmetical operations are totally different processes from the intellectual apprehension and description of the properties of an object.

It is as a result of this fundamental distinction, and not because there are separate centres for letters and figures, that the remarkable difference is observed. I am disposed to conclude that numerical operations take place at a lower level than the mental processes which result in speech. The cell-groups representing numerals may thus be accessible to the right hemisphere. There is no evidence pointing to the conclusion that numbers are especially relegated to the right hemisphere, no case has been reported in which a lesion of this hemisphere has had any effect on numbers corresponding with the effect on speech of lesions of the left hemisphere.

I have shown elsewhere that words as motor processes, which they are from one point of view, parrot-like reproductions of articulate sounds, must be organized bilaterally in the bulb and central ganglia, all the muscles concerned in speech being bilaterally associated, the word cell-groups being ready for employment by the higher centres, and, in my opinion, this bilateral organization

extends as high as the third frontal convolutions. Word cell-groups exist in the right third frontal as well as in the left, and are habitually employed in speaking in association with the left third frontal, the two being connected by commissural fibres of the corpus callosom. It is through these word groups on the right side that aphasia is obviated in some cases of right hemiplegia with marked paralysis of the face and tongue, the lesion having taken place in the white fibres of the internal capsule sufficiently low down to leave intact the commissural fibres of the corpus callosum between the frontal convolutions of the two hemispheres.

In support of this view may be cited the remarkable phenomenon of mirror writing. Some people can write currently with the left hand, but the writing is not only backwards from right to left, but reversed, so that a mirror is required in order to interpret it, when it is found to resemble the ordinary handwriting. Here the graphic centres must be bilaterally associated, and the left must have educated the right.

I may be permitted here to recall the fact that before the identification of the sensory and motor cortical areas by Hitzig and Ferrier I had inferred that they would be found in the Sylvian, Rolandic, calcarine, and occipital convolutions, since radiating fibres from the bulb and central ganglia were distributed only to those convolutions, the intermediate superadded convolutions constituting much the largest area of cortex receiving no radiating fibres.[1] This structural arrangement, made out by tracing the fibres, has been confirmed by Flechsig on developmental grounds. I have some right, therefore, to assume that other conclusions at which I arrived are valid. The most important of these was that the fibres of the corpus callosum and anterior commissure were distributed to exactly the same convolutions as the radiating fibres, and constituted a commissure between corresponding gyri in the two hemispheres. I found also a large longitudinal commissural system.

[1] Extract from "The Structure of the Cerebral Hemispheres." *Journal of Mental Science*, April, 1870:—

"It has further been found that throughout the hemisphere the distribution of the central and callosal fibres is to the margin of the respective lobes, leaving extensive intermediate tracts of convolutions, which receive no fibres from either crus, central ganglia, or corpus callosum. It is at once obvious that these superadded convolutions will be the convolutions most characteristic of the human brain, and will constitute the difference between one brain and another. (Confirmation of this is readily found in a comparison of the hemisphere of the monkey tribe with that of man.) The sensations transmitted upwards from the several sense-centres must first impinge upon those parts of the surface grey matter in which the fibres from the sensory tract or ganglia end; so, again, wherever volitions may be originated, the downward starting point of the motor impulse must be in some convolutions connected by fibres with the motor ganglion or tract."

A LECTURE ON EPILEPSY

Delivered at the Medical Graduates' College and Polyclinic

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Epilepsy is a disease of which we know extremely little definitely, and with certainty nothing at all. We are up to a certain point agreed as to what cases are to be included under the term ; we have much real and trustworthy knowledge as to the etiology of the disease ; but its pathology is largely conjectural, and its treatment mainly empirical.

Symptoms.—Epilepsy is characterized by recurrent attacks of general convulsion, attended with or preceded by loss of consciousness, usually sudden. This sudden loss of consciousness is perhaps the most constant feature of an epileptic fit. The convulsions vary in character, intensity, and duration, and may in petit mal be represented only by fixation of the eyes and features, with, perhaps, dilatation of the pupils, or by slight twitching of some of the facial muscles or of the fingers.

In true epilepsy these recurrent convulsions constitute the disease. The patient in the intervals enjoys such health as is natural to his constitution, and there are no symptoms suggestive of any affection of the nervous system. When we can refer the convulsions to a definite lesion of the cranial bones, or of the membranes or cortex of the brain, we are only too glad to remove the case from the category of epilepsy.

In a typical attack there is at the onset a peculiar cry, ending, when I have heard it, in a long expiratory groan, sudden pallor, loss of consciousness, and general tonic spasm of all the muscles, of which the groan is an expression. With the loss of consciousness is a headlong fall, sometimes in a constant direction in each attack to one or other side, backwards or forwards, or preceded by certain movements, as, for instance, by a pirouette of three turns in a case in which I saw several fits, sometimes determined by the position in which the patient happens to be at the moment. The rigidity may last till the face becomes livid. It is followed by clonic spasms, usually at first one-sided, the face being distorted, the mouth drawn towards the convulsed side, the head and eyes jerked by a succession

of spasms in the same direction, the jaw champing and froth issuing from the mouth, which may be bloody, from the tongue, also in convulsive motion, being caught between the teeth.

Respiration is now laboured, noisy and stertorous, sometimes exhibiting the nasal stertor, to which attention was originally called by Dr. Bowles. The countenance becomes purple and swollen, and the conjunctivae injected. Similar clonic convulsions affect the other side, and the fit terminates in a series of synchronous general muscular spasms, the limbs being in an extended position. Minute petechiae may be left behind as a result of the intense congestion. The urine is often passed and sometimes the faeces, at what period of the attack I do not know. The convulsions, as they subside, leave the patient in a profound stupor, at first amounting to coma, gradually shading off into sleep, from which the patient wakes up usually aching and sore from the muscular exertion, sometimes headachy, confused, and stupid ; sometimes much as after a night's rest and with no recollection of the attack.

After petit mal, which may consist merely of a momentary suspension of consciousness, with fixation of the features and staring eyes, there is no coma, but there may be maniacal excitement of extreme violence. Homicide has not unfrequently been committed in paroxysms of epileptic mania.

There may be variations in any part of the picture without any special significance attaching to the character of the fit. The severity of the case is estimated rather by the frequency than by the violence of the attacks, more especially as regards the effects on the mental faculties.

The epileptic fit is often preceded or initiated by what has been called an aura, an odour, a flash of red or coloured light, a subjective noise, a sensation in the epigastrium or in one of the limbs travelling towards the heart ; a comparison of the sensation to a breath of wind suggested the name of aura. The starting point of the aura has been supposed to indicate the part of the brain in which the initial disturbance occurs.

An epileptic fit may come on at any period of the twenty-four hours, and may seem to be determined by a great variety of circumstances. A favourite time for the occurrence of attacks is in the night, especially towards morning, and again soon after rising. A patient may suffer from nocturnal epilepsy for years without knowing it, and when a case first comes under investigation it is always important to inquire if the patient has been subject to bad dreams, has ever wet the bed, or has found the bedclothes or himself on the floor. There are cases in which the seizures take place at no other time. It is important to note

that they rarely come on during exertion or excitement. We need not forbid epileptics to take exercise or to ride, or even to cycle in moderation. In this conclusion I am glad to have the concurrence of my friend, Dr. Buzzard.

Causation.—In the causation of epilepsy the most important element is undoubtedly an inherent tendency in the nervous system. Epilepsy runs in families with other neuroses. It is common in idiots and often associated with a low type of nervous organization, intellectual and moral, and with a misshapen head or some bodily defect or deformity.

This inherent tendency or predisposition is for the most part congenital, but it varies greatly in degree. In some cases it is so powerful that the attacks occur early in life, and in spite of every care and of the most favourable conditions; in others they take place only under the influence of some violent exciting cause, or when the general health has run down or the nervous system has been exposed to some deteriorating influence such as alcoholic excess, immoderate smoking, or sexual abuse. If we are to call by the name of epilepsy those cases in which recurrent attacks of convulsions are distinctly due to an obvious peripheral irritation ceasing on its removal, we must admit that epilepsy may exist without any inherent tendency in the nervous system properly speaking, other than such as may be generated by peripheral irritation. On the other hand, it is possible that a predisposition may lurk in the nerve centres, and never be brought out in the course of a long life.

I am not sure that sufficient importance has been assigned to the sensory nerves in the causation of epilepsy. It is more and more recognized that the sensory side of the nervous system is the dominant one. Sensory impressions are the initial nervous operations, all motor actions are in response to afferent impressions, and the sensations to which they give rise constitute the primary basis of all intellectual processes. We look upon the brain and nerve centres as the structures in which nerve force or energy is generated; but whatever may be the nature of nerve force, it must be formed and liberated in the sensory nerve endings, or, rather, beginnings. The impulse here originated, which travels to the spinal cord and brain, and starts reflex action or is transmuted into a sensation, is as much a manifestation of nerve energy as the impulse which, coming down from the spinal cord, excites muscular action.

Nothing can be more clearly established by experience than the relation between painful dentition and convulsions in childhood. Convulsions which have been going on for hours may at once cease when the tense and painful gums are incised. Gastro-intestinal

derangement, again, is a frequent cause of infantile convulsions. Cases also are related in which the expulsion of a tapeworm or of long round worms has been followed by the cessation of convulsions.

It is a generally accepted view that the sense of buoyant health or its opposite—a feeling of unaccountable depression—is dependent upon the impressions transmitted to the nerve centres from the great viscera, and it is reasonable to infer that since peripheral irritation, such as the instances just given, can give rise to recurrent convulsions, a persistent visceral derangement may possibly be a cause of epilepsy.

It is, perhaps, worth while referring to the remarkable fact that an epileptic fit can sometimes be averted, and even an incipient Jacksonian convulsion arrested, by a tight band bound round the wrist or arm. If a peripheral impression can produce such an effect in the way of inhibition, it may be inferred that impressions of another kind and degree are capable of generating a tendency.

While the tendency to epilepsy must be looked upon as chiefly inherited, it is not very commonly manifested in infancy or childhood. Cases occur in which the disease appears to be established by infantile convulsions, but infantile convulsions must not be regarded as constituting an epileptic tendency, since they may happen to children entirely free from neurotic inheritance, and who never subsequently suffer from nervous affections.

When infantile convulsions are followed by epilepsy it is probable that the brain has been damaged.

A noteworthy point in the clinical history of epilepsy is the frequency with which the fits begin to occur at puberty or during adolescence. So close is the association between epilepsy and this period of life, that when the first attack is much deferred and takes place in the adult, we look for some contributory cause which has brought out the latent inherited tendency, or suspect that we are dealing not with epilepsy as such, but with some morbid condition of which epileptiform convulsions are among the symptoms, syphilis in the young adult, cardio-vascular derangement or degeneration after middle age. It is not necessary to dwell on the various causes of nervous stress and excitement incident to early adolescence.

When epileptiform convulsions set in suddenly and frequently at any period of life peripheral irritation of some kind may be suspected. One such instance in my experience was a necrosed terminal phalanx, the fits, previously frequent and violent, ceasing when the bone was removed. Another was a piece of glass embedded in the wrist in close relation with the median nerve; pressure on the spot at once brought on a convulsion.

Pathology.—In the absence of definite knowledge as to the causation of epilepsy, we fall back upon every kind of speculation and consideration which may throw light upon the problem which the disease presents. If we only knew completely and exactly what takes place in the nervous system when a convulsion occurs, we should have gained a step towards the comprehension of a disease, the most prominent feature of which is recurrent convulsions, though not perhaps any great advance, since a convulsion is not the first, possibly not even the most important, element in an epileptic attack.

A convulsion is the result of an excessive and disorderly liberation of nerve energy, as an ordinary purposive action is the result of a duly limited and orderly nervous discharge. This nerve force is generated in and by nerve cells or by reaction between the cells and the intercellular plasma; in any case, it is through the cells and their axons that the energy is conveyed to the muscles.

We do not know the precise nature of this force, but it can only be a transformation of forms of energy with which we are more familiar, and everything points to a chemical change of the nature of oxidation as its source. From the fact, indeed, that oxygen seems to be absolutely necessary to the functional activity of the cerebral cortex, it would almost seem that here the oxidation was direct, but such is not the case in the same degree in the grey substance of the spinal cord.

Another point which we may infer with regard to nerve force is that it must be characterized rather by intensity than by quantity; its electrical analogue will be the spark of static electricity rather than the galvanic current. The chemist cannot point with confidence to the products of oxidation of the nervous structures and estimate the amount of waste they have undergone from the exercise of their functional activity, as he can in the case of muscle.

Another consideration pointing in the same direction is that there must be some quantitative as well as qualitative relation between the chemical process by which nerve force is set free and the substances by which this process is influenced. It is not simply by its presence in the blood that hydrocyanic acid at once arrests the evolution of nerve force in the vital centres of the medulla, or that morphine more gradually suspends the functional activity of the cortex, and ultimately of the lower centres. A fatal dose of prussic acid being equally distributed in the blood, only an incalculably minute proportion can be carried to the medulla; and in whatever way it interferes with the chemical changes by means of which the nerve force is evolved, it affords a means of estimating the amount

of chemical change and of showing that quantitatively considered it must be extremely small.

From whatever point of view the evolution of nerve energy is considered, the conclusion suggested is that in the grey matter there is accumulated a substance of an explosive character ready to liberate energy, not by gradual combustion like coal, but by a sudden readjustment of chemical affinities, as in the case of nitro-glycerine.

The passage of an electric spark through a mixture of oxygen and hydrogen will give rise to a violent explosion. The gradual burning of like volumes of one gas in the other will liberate an equal amount of energy, but gradually. An explosive compound such as nitro-glycerine may be said to consist of carbon, hydrogen, and oxygen in explosive proportions, held apart by combinations into which nitrogen enters, which defeat their natural affinities. Let any condition be applied which destroys this restraint—flame in the case of gunpowder, percussion in the case of gun-cotton—and the normal affinities assert themselves with explosive violence. A piece of gun-cotton will burn harmlessly on the open hand; fired by a percussion cap it would blow the hand to pieces.

A convulsion, then, represents an explosion of nerve matter—that is, a more or less general discharge of the explosive material accumulated in the grey substance of the cortex of the cerebrum or cerebellum or of the central ganglia. A question which at once arises is how such discharge is determined; and here experiment gives us the usually contradictory suggestions, while clinically it occurs under the most diverse conditions.

Convulsions attend death from loss of blood and from suffocation; we may have them at the invasion of almost any of the acute specific fevers, and they are common in disease of the kidneys; they may mark the onset of acute inflammatory affections of the brain, or occur at almost any period during their course, or as part of the final symptoms. Convulsions, again, may be excited by peripheral irritation—as in teething of children already referred to. It is difficult to draw trustworthy inferences from such diverse premises, and there is not time for any such discussion as might serve to establish even provisional explanations. It may be stated, however, that the experimental methods by which convulsions are induced have in common arrest, more or less sudden, of the cerebral circulation. It will be remembered that Hughlings Jackson attributed convulsions to spasm of the cortical arterioles; and I may, perhaps, add that for many years I have held the view that uraemic convulsions are due to stoppage of the circulation in the cortical capillaries, and not to the direct action of any poison.

There remain the definite indications of Jacksonian epilepsy and of the experiments of Hitzig and Ferrier. There can be no question that electrical stimulation of the cerebral cortex and of various subcortical grey centres can excite muscular actions, local and general, and nothing is better established than the causation of convulsive movements of a part by a lesion of a cortical centre. A local convulsion traceable to such a lesion may also become generalized with loss of consciousness.

The explanation of epilepsy to which the foregoing considerations lead up is that the explosive material in the cortical grey matter or other important collections of grey substance is unstable, and goes off too easily. It is this inherent instability—the liability to explosion without adequate exciting cause, pehaps even spontaneously—which characterizes epilepsy and distinguishes the epileptic convulsions from convulsions determined by some violent external cause.

It is sometimes assumed that a convulsion exhausts the explosible matter in the nerve centres, and Dr. Hughlings Jackson has spoken of the coma which follows an epileptic seizure as paralysis from such exhaustion. This view seems to me to be disproved by the status epilepticus where one convulsion succeeds another for many hours with intervals of unconsciousness.

The periodical recurrence of the fits at almost regular intervals, irrespective of any apparent provocation, is suggestive of accumulation of the explosive nerve substance up to an amount beyond the control of the normal inhibitory influence. It might be, on the one hand, defective constitution of the nervous material, or, on the other hand, imperfect inhibitory control.

Mostly it is an inferior quality of nerve substance which is formed, and the instability is evidently a result of malnutrition. It is conceivable, however, that it may not be the formation of a low grade of nerve substance, but of too sensitive a type of explosive material. Possibly in this we have some sort of explanation of the undoubted fact that men of genius—military, literary, and scientific—have been epileptics.

Night and Early Morning Attack.—It has already been mentioned that the fits of epilepsy are extremely liable to come on during the night or in the early morning or soon after rising. This is highly suggestive of cardio-vascular conditions as among the the influences by which the time of an outbreak is determined. It is at about 4 a.m. that the temperature of the body is lowest, during sleep the circulation seems to run down and the vascular pressure to fall. In cyclic albuminuria, which is most common in adolescents, it is the change from the recumbent to the erect position

(with perhaps exposure of the skin) that determines the appearance of albumen in the urine, and if such change of posture can so far affect the circulation in the kidneys, it may influence in an even greater degree the circulation in the brain. This fact is at any rate evidence of the lowered tone of the cardio-vascular system during sleep.

Prognosis.—The prognosis in a case of epilepsy turns mainly on the question how far it is due to a tendency inherent in the nervous system, how far to causes outside the nervous system which might either generate or aggravate the tendency, or, the tendency being there, provoke attacks.

Heredity will enter very powerfully into the consideration. It is not necessary, however, in order to come to an unfavourable conclusion, that there should be a family history of epilepsy. Insanity, alcoholism, marked eccentricity, vice, perhaps even hysteria or migraine in parents or near relations, may be evidence of hereditary instability.

If, with the liability to recurrent attacks of convulsion, there are other evidences of a low type of nervous organization, such as idiocy or weakness of intellect, or moral depravity; if there is any faulty construction of the cranium, if the head is too large or too small, or if there is the expression which we recognize and call epileptic, there is practically no chance of the cessation of the attacks.

If, again, the fits have begun in childhood, or at or soon after puberty, and have recurred regularly and frequently, whether or not there have been other injurious influences at work, it may be concluded that an inherent tendency to instability exists.

If, on the other hand, the patient has the look of bodily and mental vigour, if some serious exciting cause has preceded each attack, if the fits have not begun till late in the period of adolescence or till adult age has been reached, it may be presumed that the inherent tendency to epilepsy is not strong, and that it may possibly be held in abeyance.

Very important indications also may be furnished by the state of the circulation. Prolonged observation has convinced me that the tension of the pulse is unduly low in epileptics. This low-tension pulse I have come to consider as a characteristic of the disease. It is a part of the general sluggishness of the organic operations like the pallor which is common and the dull complexion and flabby skin. Whether the deficient arterial pressure is a cause of the malnutrition of the nervous centres, or a mere consequence, its significance is the same and is an unfavourable prognostic indication. A pulse of unduly high tension for the age of the patient,

on the contrary, is a ground for hope of recovery. More than twenty years ago a boy, the son of an army medical officer, who had had to leave the navy on account of epileptic fits, was brought to me. He had a high-tension pulse and exhibited no signs of bodily or mental weakness. I ventured to give a favourable opinion as to his future, and when a year or two had passed without a fit, his father's influence secured permission to enter the army. I met this patient accidentally last year in excellent health. He had only had one attack after he joined the army; this was during one of the Afghan campaigns under circumstances of great fatigue and privations, and it did not interfere with the discharge of his duties.

Treatment.—In all cases the epileptic should have an open-air life as far as possible, and should be provided with employment in which he can take an interest and occupy his time. Nothing could be more satisfactory than the experience of the Epileptic Colony, near Chalfont, and in the much larger establishments in Germany and America.

Exercise of almost every kind may be permitted. Attacks rarely come on during exertion, and I have not hesitated to allow patients to ride. I am not quite so confident with regard to cycling, though I have never heard of an accident attributable to a fit, and I should only permit of swimming under competent supervision. The fear with respect to games is not of an attack at the time, but during the reaction afterwards and especially next morning.

A very important question is whether an epileptic boy or girl should be allowed to go to school. Unless the fits were frequent I should answer in the affirmative, particularly as regards boys. The grounds on which hesitation would arise would be the effects on other children of witnessing a fit and the dread of its being known that the child was an epileptic. Both considerations would apply more seriously to the case of a girl; it would tell more against her in after-life, and girls are more susceptible to violent nervous impressions than boys.

Epileptic children and young people should be very carefully dieted, but not, so far as my opinion goes, on any particular or peculiar principle. The food must be adjusted to the digestion of the individual, the object to be held in view being to keep the general health at its best. Overfeeding should be strictly avoided, and the evening meal should always be light. It must be borne in mind that constipation or flatulence may bring on a fit, and the wholesome neglect of trifling derangements of the digestion to be recommended in healthy adolescents is not permissible when dealing with such as are subject to epilepsy. No stimulants

should be allowed. It is doubtful whether they are ever of any service; and there is always the danger that the unstable nervous organization may fall under the influence of alcohol.

The first point to be determined will be how far the case is one in which the inherited tendency is dominant and in which palliation only is to be looked for, or whether a hope of complete recovery may be entertained. But even in the most hopeless cases we should study the general constitutional conditions and the functional efficiency of the different organs. Exciting causes should as far as possible be identified. In one constipation may be found to determine an attack, in another imprudence in diet, indigestible food, heavy or late meals. The menstrual period may be attended with attacks before, during, or after, even when quite normal; still more when menstruation is irregular or attended with severe pain is it liable to provoke fits.

The treatment of epilepsy, for the most part, resolves itself into the routine administration of bromides, just as the mere discovery of a cardiac murmur is too often the signal for giving digitalis or strophanthus. Whether the attacks are severe or slight, attended with violent convulsion or consisting of a momentary suspension of consciousness, whether the intervals are long or short, the rule is to give bromides. The potassium bromide is the most efficacious but it is depressing, and ammonium, sodium, and of late years strontium bromide, are had recourse to, singly, or more frequently in combination.

It is not to be denied that the bromides have a marked influence in reducing the frequency and violence of the attacks, or that it is a great gain to the sufferer, but in no disease is it more true than in epilepsy that to relieve symptoms is not necessarily to cure the disease; and the question arises whether the relief of symptoms, the diminished frequency and mitigated violence of the fits, may not in many instances be too dearly purchased. That such is the case is distinctly my opinion. An almost more important consideration even than the depressing effect on the sufferers themselves is that the ready routine method of dealing with epilepsy interferes most seriously with the study of individual cases.

We are familiar with the bromide rash, and an attempt is generally made to obviate its eruption by arsenic. Less conspicuous and attracting less attention, though infinitely more important, is the lowered tone of the nervous system, the loss of energy and memory, the lessened efficiency, the depression of spirits and impaired enjoyment of life. I have seen patients reduced to a condition of practical dementia by long-continued

dosing with bromides, and when the attacks have ceased it has seemed to be because the nerve centres had no longer energy sufficient for an attack. Julius Caesar and Napoleon are said to have been epileptics; I am quite certain they would not have won their battles or left such a mark in the world's history if they had been energetically treated by bromides. It may be added that large doses of bromides have been found experimentally to affect unfavourably the dendrons and gemmules of the cortical cells.

A very important question is here suggested: what is the ultimate effect of postponing the fits? One view is that each attack predisposes to others, diminishes the resistance to the outlet of nervous energy along certain channels, tends to the establishment of a habit. Some sort of support is lent to this view by cases in which the fits tend to occur in groups or series; but there can be nothing of the kind in cases in which a single attack takes place at long intervals, say of months, and, according to my judgment, a general survey of the disease indicates not so much a liability to the outburst of nerve energy from deficient resistance along certain paths as to the formation of an unstable nerve substance by malnutrition. What we have to aim at is not to dull the sensitiveness of the nerve centres, but to increase their vigour and stability.

The idea on which I base my practice in regard to the treatment of epilepsy is that the use of the bromides is to diminish the frequency and severity of the fits, while the cure of the disease, the removal of the instability of the nervous system, is to be sought by other means. Occasionally the instability of the explosive nerve substance may be due to some visceral irritation, especially perhaps ovarian, and protection from this by the diminished sensitiveness to external impressions which is produced by the bromides may allow the nerve centres to regain their equilibrium, or give time for the subsidence or removal of the disturbing influence. It is also possible that if a brain had not originally any considerable inherent epileptic tendency, but had acquired the liability from some of the varied influences which lower the nervous tone, the prevention of the fits might afford time for the repair of the injury done by the adverse conditions. But for the most part there will be functional derangements or states of imperfect nutrition which may be dealt with in the intervals. I think there is a possibility of more harm being done by the bromides in the intervals by a deteriorating influence on the brain than the mere postponement of the attacks would be worth.

The rules, then, which I lay down for myself are somewhat as follows: In what I call long distance epilepsies, in which the intervals between the fits are six weeks or over, I do not give bromides

regularly and continuously unless there is some other indication for their employment, such as sleeplessness or excitement. If the attacks recur punctually at definite intervals I should try to avert them by a short course of bromides when they were due. In any case, if the patient has premonitory symptoms which he recognizes, he should be instructed to take bromides as soon as they come on. If, again, he is aware that an attack is liable to follow any special incident in his life, exertion, excitement, worry, such social events as a concert, theatre going, or dinner party, he may try to baulk it by bromides.

When the fits are frequent, and especially when it is clear that the bromides exercise a restraining influence, they must be given in such amount as may be found necessary and at such hours as may be suggested by the time at which the fits are liable to occur. In particular a dose will be given at bedtime when the attacks come on in the night or early morning. Even here, however, the time gained between the fits should as far as possible be turned to profit. We should not rest content with the diminution of the attacks, but should endeavour to discover and remove the underlying cause.

It appears to me that there is an opening for fruitful inquiry in those cases in which the attacks are nocturnal, and again in those in which they occur soon after rising. Clearly here there is cardio-vascular instability which reacts upon the nervous instability, and it would seem to be a reasonable anticipation that by moderating the running down of the vascular tone which takes place during the night the liability to attacks would be diminished. This is a course of observation which can only be carried out by the family medical man or by the resident officers of an institution. A tea-cup of hot and strong beef tea might be given at bedtime, or a glass of hot milk. In the case of morning attacks this might be taken immediately on waking, and at this time cayenne or pepper might be added to the beef tea, or perhaps a little spirit of ammonia or something pungent to the milk. I should infinitely prefer food to drugs ; but, food failing, a dose of digitalis or other cardio-vascular tonic at night ; in the morning, perhaps, nitro-gylcerine might be given.

In seeking to apply remedial measures, as distinct from the prevention of fits, we must first inquire carefully into the patient's habits and mode of life, and put a stop to anything which is adverse to health, or especially which may exhaust the nervous system. Irregular meals, late hours, sedentary habits, self-indulgence of every kind, especially such as is vicious. Discipline is of the greatest value. The genuine epileptic is usually deficient in will power, and

needs the outside help which is comprised in the term discipline. But I need not dwell on these general measures, though they are of great importance.

We must next ascertain whether there are any persistent functional derangements, and a thorough physical examination of the chest and abdomen ought to be made. Constipation must not be permitted, still less loose and irregular action of the bowels. Indigestion, especially when associated with flatulence, must be corrected. All abdominal uneasiness must be looked upon with suspicion, as must any irregular distribution of the gases in the stomach and intestines, or general distension of the intestinal canal. The urine will of course be examined, and not merely for albumen and sugar, but as affording evidence as to the general condition of the patient.

Disorders of menstruation may play an important part in epilepsy. It does not follow that because the fits in any given case occur chiefly at the menstrual period, which is otherwise normal, the epilepsy is of ovarian or uterine origin, but, on the other hand, ovarian irritation or dysmenorrhoea or other derangement may be responsible for attacks taking place at any time. I cannot of course go into the treatment required by this class of cases.

There remains the question whether anything can be done to improve the stability of the nervous centres by such remedies as are known to be of service when the nervous tone has been impaired, or when the functional activity of the brain has been disturbed by conditions of the circulation.

I have already stated that in epilepsy generally the pulse is small and the arterial tension low. Exceptions occur, and in my experience, when a young epileptic has a pulse of unduly high tension, the prognosis is favourable. The tension can be brought down by care in diet, and by eliminants. In post-hemiplegic epilepsy, again, when the pressure within the arterial system, which has resulted in the rupture of a cerebral vessel, persists, the frequency of the fits can be materially reduced—I think I have seen them kept off altogether—by keeping down the pulse tension, which can best be effected by mild mercurial aperients.

For the generally-lowered vitality of epileptics I have given phosphorus and the hypophosphites, or arsenic with strychnine or quinine, and sometimes iron. I am disposed to think that the arsenic, which is often given at the same time with the bromides to prevent the rash, may sometimes have a considerable share in the production of good results. I cannot say definitely that I have seen cures, because in consulting-room practice we cannot follow up patients who recover, but I have seen many instances of remark-

able improvement, the fits becoming less frequent and severe month by month. A satisfactory feature of such treatment has been the increased sense of well-being and enjoyment of life which has attended it, the improvement of the memory, and generally increased intellectual efficiency. This has been particularly noteworthy when bromides which had been taken for some time were left off.

Epilepsy will repay further study. Jacksonian convulsions have been differentiated from true epilepsy; the recurrent convulsions caused by local or general thickening of the cranial bone of traumatic or idiopathic origin ought to form a distinct category, and I think that further distinctions will be established so that we shall speak of the epilepsies rather than of epilepsy. The different forms will then have each its own treatment. It is because my thoughts have moved in this direction that I have brought the subject before you.

A CASE OF HYSTERICAL ANAESTHESIA OF BOTH LEGS BELOW THE KNEE. TREATMENT BY METALLIC BANDS: CURE

Clinical Society's Transactions, 1878

SARAH G., housemaid, aged 17, was admitted into St. Mary's Hospital, July 2, with impairment of muscular power in both upper extremities, and anaesthesia of both legs and feet.

Catamenia commenced two years ago. Until this time her health had been very good. Since this she has suffered much at her periods from headache, pain in back and in left side; sometimes also in right. Menstruation has been regular every three weeks, lasting about three days, and normal as to quantity. During last four months pain has been persistent in forehead, lower part of back, and in left side of abdomen. She has been obliged to give up her occupation, being very weak and fainting after exertion. Loss of muscular power and sensation have come on gradually. Has never had hysteria.

Present condition.—Considerable loss of muscular power in both arms and hands. No loss of sensation. In both legs from the knees downwards there is total loss of sensation. No evident loss of muscular power. Above knees sensation is not impaired. She can raise both arms nearly up to head, but can scarcely squeeze at all with the hands. She can get out of bed and walk about, and also stand with feet close together and eyes shut; yet there is no sensibility in feet or legs. When either leg is pricked with a needle, patient's eyes being shut, she does not know what is done. A needle is even thrust down under either great toenail, but not the slightest indication of pain is given. Patient says that if she could not see, she should not know that she had legs and feet. In all other parts of the body sensibility is normal. Both legs and feet are very cold; when pricked no blood is noticed to exude. Whole body is well nourished, and has not an anaemic appearance. Pain is felt on pressure being made in either ovarian region, but most in left. Senses of smell and taste normal. Sight varies; sometimes can see well, at others two or three things are seen when she is looking only at one. Nothing abnormal noticed about optic discs. Other organs normal. Temperature normal.

Treatment.—Ordered pil. galbani co. gr. 10 twice a day. General health improved somewhat, but no other change took place.

July 11. Metallic band ordered to be placed round right leg.

On the 12th patient said there was a slight feeling of weight in the right leg, but she could not feel the band round it, nor did she feel when pricked.

13th. She thought she could feel there was a something round the leg, but there was no decided return of sensibility.

15th. When the leg was sharply pinched patient said she could feel it. When her eyes were shut and this leg was pricked anywhere, she could mention the place where it was done. There was no feeling of weight in the leg, but some tingling when walked upon.

17th. Patient could feel as well as ever in this leg and foot, but the other remained as devoid of sensibility as before. This was proved by pricking and thrusting a needle again under the toe-nail, when no pain whatever was evinced. The band was still kept on for some days, then transferred to the left leg, in which sensibility returned in much the same way as in the other leg, but more quickly. Muscular power improved in both arms; she gradually lost headache and other pains. She was discharged on August 2, with full sensibility in each leg and foot, fair muscular power in both upper extremities, and in good general health.

The treatment adopted in this case was, of course, suggested by the results of the application of plates of gold and other metals in hysterical hemianaesthesia, as described by Professor Charcot. His experiments, which were made the subject of a report to the Academy of Medicine, were undertaken at the instance of M. Burck, for the purpose of testing his views as to the therapeutic influence of gold in certain affections of the nervous system. The patients, hysterical girls or women, had pain or tenderness in one or other ovary, more commonly the left, and the corresponding half of the body had completely lost sensation. A large pin could be thrust through the skin at any part of the trunk or limbs without being felt, and without the patient knowing that anything had been done, while no blood issued from the punctures unless a vein had been pierced. A band, in which plates of gold were secured, was now placed round the arm, the gold in contact with the skin, and in fifteen or twenty minutes sensation had returned, and the punctures bled; the return of sensation taking place first near the band and gradually spreading upwards and downwards from this part. According to the experience of the Salpêtrière, it was not a matter of indifference what metal was employed. Gold was most generally efficacious, but in a few cases it failed, and some other metal had the power of inducing a return of sensation. It was found that a

feeble galvanic current was set up by the contact of the metal with the skin, and it has been suggested that this was the curative influence.

I may here mention a fact which struck me as very remarkable when a number of these cases were shown to me by Professor Charcot, during a recent visit to the Salpêtrière, viz. that the subjects of this hemianaesthesia were some of them engaged in sewing, and appeared to have no difficulty in holding the work or even the needle. It did not occur to me at the moment to suggest the application of a test I often employ, which is to ask the patient to pick up a pin from a smooth table blindfold; but apparently the women were not under the necessity of keeping their eyes on the work, or on the objects held in the hands.

In the case related no care was taken in the selection of the metal, and brass was employed. It is clear that the band was in some way or other instrumental in the recovery of sensation, and that it was not due simply to rest, food, the moral influence of residence in the hospital, and change of surroundings, since feeling returned only in the leg on which the metal was worn. It is probable that as brass is more oxidisable than gold there would be more powerful galvanic currents, but no test was applied, and I attribute the improvement in this case simply to expectant attention. The girl looked for the result, her attention was constantly called to the part by the presence of the band, and this was sufficient to produce the effect. I was careful not to express any confident anticipation of a cure to the patient, or to the students or nurses in her hearing, though this artifice would have been justifiable, and would probably have had considerable influence, as I wished to make the experiment without resort to such measures.

The absence of haemorrhage from punctures is a remarkable feature in these cases. I am disposed to attribute the anaesthesia to the condition of the capillary circulation indicated by this, rather than directly to changes in the nerve-centres.

EXTRACT FROM A PAPER ON THE CAUSATION AND SIGNIFICANCE OF THE CHOKED DISC IN INTRA-CRANIAL DISEASES

British Medical Journal, 1872[1]

After discussing the theories of the causation of the choked disc, he relates a case of tubercular meningitis and post-mortem findings, finishing thus :—

The nerves, followed to the back of the eye, exhibited large superficial vessels quite to the eyeball, close to which they expanded into a bulbous enlargement, the constriction at the nerve-entry being very abrupt. The swelling had not a translucent appearance, and it did not disappear on slight traction or pressure. The optic discs were not very well defined ; there was no elevation. The retinal vessels were distinct—arteries as well as veins.

(A second case is related, and he continues) :—

The optic commissure and tracts were imbedded in solid yellow exudation, but apparently normal when extracted. The right optic nerve near the eye was expanded into a bulb of considerable size, evidently formed by distension of the outer sheath by fluid ; it was quite translucent. The disc was fairly defined, not papillated, not so pale as usual. The retinal vein was extremely large and distinct, issuing from the centre of the disc. The left optic nerve had no bulbous expansion, and no appearance of having been distended. Longitudinal vessels of considerable size were visible on its exterior. The disc was not well defined, but had a sort of halo round it. The retinal veins were rather large ; the arteries small, but distinctly visible. Both vessels were concealed in the disc and for a short distance beyond its margin, apparently by exudation. It is noteworthy that, in this case, the distension of the nerve-sheath, usually bilateral, was present on one side only ; and this was not due to accidental displacement of the fluid on the other. The difference in this respect coincided also with a marked difference in the degree of change in the intra-ocular termination of the nerve, the congestive or inflammatory process having gone further in the eye, the nerve-sheath of which was not distended.

It appears to be a disputed point, whether the fluid in the vagina

nervi is formed on the spot, or is forced along the lymph-space from the arachnoid cavity. It has been shown that injections can be made to pass from the arachnoid space into the nerve-sheath; and in my opinion it is in this way that the sheath is distended in these cases of meningitis. The fluid and the pressure are present; and it is unnecessary to seek another cause for an effect which they are proved to be competent to produce. On this view the dropsy of the nerve-sheath is simply another example of intra-cranial pressure, producing its effects in the direction of least resistance, tending to confirm this hypothesis as to the mode of production of the choked disc, and affording an illustration of the difference in the results of pressure having different sources of origin.

According to my experience, while some degree of hyperaemia of the disc is common in tubercular meningitis, well marked choking of the disc is rare; and I have never seen an extreme example, such as I have met with in cases of cerebral tumour. If my experience on this point be borne out by that of other observers, and if, as I expect, hydrops vaginae nervi be found to be much more common in tubercular meningitis than has been supposed, the occurrence of marked congestion and swelling of the disc in cases of cerebral tumour, and the less frequent and less pronounced occurrence of this condition in tubercular meningitis, in which the general intra-cranial pressure is apparently at least as great, would be capable of a satisfactory explanation.

Tumours are not usually accompanied by much fluid in the arachnoid cavity; and the increased pressure will act in the obstruction of return of blood from the orbit into the cranial cavity, while at the same time the force required to carry the blood through the vessels of the brain when subjected to pressure would tend to distend the capillaries of the disc, on which no adequate counter-pressure is brought to bear. On the other hand, in tubercular meningitis the fluid in the arachnoid cavity is, at any rate, one source of pressure; and it is to be expected that it will find its way along the sheath of the nerve, and distend it at its weakest part. This fluid pressure also, though it will obstruct the return of venous blood from the orbit, will compress the arteries carrying blood to the eye; but more particularly it seems to me that the compression exercised along the whole length of the nerve by the fluid within the nerve-sheath would have the effect of neutralizing in some degree the transmission of pressure along the minute vessels of the nerve from the cranial cavity, and so tend to diminish the force brought to bear on the vessels of its intra-ocular termination.

REMARKS IN DISCUSSION ON DR. C. MERCIER'S PAPER ON INHIBITION

Brain, 1889

DR. BROADBENT, after some preliminary remarks, said :—

Like Dr. Jackson, I have an hypothesis as to the seat of the morbid change in paralysis agitans, which, emboldened by his example, I venture to mention ; I believe the lesion to affect the muscular nerve-endings. The idea as to the nature of nerve energy or nerve force which underlies Dr. Mercier's reasoning on the subject of inhibition, and which is generally accepted, is that each cell is a reservoir in which force is stored, and each fibre a channel by which force is carried ; the whole conception is one of liberation and transmission of energy. An impression on a sense organ sends a wave of force up to the centres ; a motor impulse is an explosion in certain cells of the cortex, and the energy set free travels downwards till it impinges upon given muscles and calls them into action.

I have for a long time held a different hypothesis of the nature of nerve force, and of the relation in which the nerve-centres stand towards each other and towards the peripheral nerve-endings. This is that the different centres among themselves, and the centres and the peripheral terminals of the nerves in muscle or sentient surfaces, hold each other in mutual tension, the periphery reacting on the centres in immediate relation with it, and the lower upon the higher centres, as well as the higher upon the lower and upon the periphery, and that a nervous impulse is not so much a transmission of energy as a disturbance of equilibrium. The lower centres and motor nerves and muscles do not passively await the shock of the explosion in cortical or other cells, but are constantly in touch with these cells by a sort of back pressure.

The best illustration of the mode of action which I believe to obtain throughout the nervous system is furnished by telegraphy. When a message is sent across the Atlantic, it is not a flash of electricity which starts from the battery here, travels along the wire and affects the needle at the other side.

There is a battery at each end, and the connecting wire is maintained in a condition of electrical tension or charge. What happens in the transmission of a signal is that disturbance of equilibrium at one end determines a corresponding disturbance of equilibrium at the other. It is in virtue of the susceptibility, due to this state of charge in the connecting wires, that multiple and opposite messages can be transmitted at the same time along the same wire. In the same way it is not force generated or liberated in the cells of the cortex, and transmitted to the muscles which sets them in action, but the entire chain of centres and the nerve terminals being in a state of tension or charge, energy liberated in certain cortical cells disturbs the equilibrium of corresponding lower centres, and ultimately of the muscular terminals, which last, as by the pulling of a trigger, set free the latent energy of the muscular fibres.

This idea of the relation between the nerve-centres and the periphery, in which, not the centres only, but the nerve-endings and the structures in which they are distributed exercise the office of batteries, which maintain the connecting nerve fibres in a state of charge, seems to me to render more easy of comprehension various phenomena normally exhibited by the nervous system, and it may, when more fully worked out, throw light on morbid phenomena. For example, it gives to the peripheral end-organs an importance contended for by many investigators and thinkers, but generally ignored. Ganglionic powers, it is true, are conceded to the retina and to the expansion of the auditory nerve, scarcely inferior to those of the cerebral cortex, but corresponding powers must belong to the cutaneous nerve-endings. Muscle, again, has been said to be only less nervous than nerve; on the view here contended for, this follows naturally, and muscular tonus is an expansion of the tension assumed to exist.

Again, the degree of tension maintained throughout the nervous system may be high or low. When it is low there will be diminished stability, and, as a result, the effects which Dr. Warner has described and represented graphically before the Society.

In telegraphy the equilibrium may be disturbed either in a positive or in a negative direction, and it is not impossible that some parallel to this may occur in the nervous system. It would follow, too, on the hypothesis of mutual resistance of the centres among themselves, upwards as well as downwards, on both the motor and sensory sides of the nervous system, that sudden withdrawal of this resistance on the part of a lower centre might determine discharge in a higher centre; in this way epileptiform convulsions might have their starting point in the basal ganglia or in the cord.

So far illustrations are taken chiefly from the motor side of the

nervous system, but, of course, the relation of tension will exist between the sensory and motor sides of the nervous system, between the sensory and motor nerve-nuclei at the same level in the cord which are concerned in simple reflex action, between the basal sensory and motor ganglia, and between associated sensory and motor areas in the cortex; between the two hemispheres, and between the cerebrum and cerebellum, and between these again and the cord.

Inhibition is a simple corollary of the relation here supposed to exist between the different parts of the nervous system. The increased knee-jerk induced by pulling the hands is an expression of the intensified general tonus, and the inhibition of reflex and other movements by powerful impressions on sensory nerves, which finds no place in Dr. Mercier's theory, is explained by the heightened tension thereby induced, primarily in the related centres, and then throughout the nervous system.

Trophic changes seem to me to be more comprehensible when the importance of the nerve terminals is recognized, and their relations with the tissues are considered. The ultimate ramifications of the nerves, in the structures to which they are distributed, are so completely lost in these structures that they may be said to form part of them, unless, indeed, we say that the structures, especially such structures as skin and muscle, are the nerve terminals, instead of saying that they receive them; they have everything in common, and whatever affects the nutrition of the one affects that of the other. On the one hand, then, the state of nutrition of the tissues will determine the degree of tension maintained in the periphery of the nervous system, and, on the other, the tension prevailing in the nerves will influence the nutritive operations in the tissues. It is interesting to note that, in the most familiar trophic derangement of nutrition, Herpes Zoster, the disturbing influence travels in the opposite direction to the normal functional impressions in the nerves affected, which are sensory and centripetal.

Another recommendation, which the view of sustained charge and mutual tension of the nerve-centres and periphery, which I have attempted to sketch, has for my mind, is that it renders somewhat less incomprehensible the extraordinary phenomena seen in hysterical women, which Charcot has brought to our knowledge. Physiology, as yet, has scarcely taken them into account, and in nervous pathology they stand apart and are not correlated with other diseases. A distinguished friend of mine indeed once went so far as to say that women ought to be excluded from the domain of nervous pathology. In my opinion, on the other hand,

the physiology and pathology of the nervous system will have to be rewritten from the point of view opened out by these phenomena. There is no structural difference between the nervous system of a hysterical subject and that of a healthy man, and the hemi-anaesthesia with its transference from one half of the body to the other, and the hypnotic phenomena, betray tendencies and reveal potentialities and relations dormant in the healthy nervous system. Now, if all parts of the nervous system are in a condition of charge and hold each other in mutual tension, the transference of hemi-anaesthesia becomes in a way comprehensible; and the comparison which makes hypnotism a state of polarization of the nervous system is in some measure justifiable. Nothing could be easier than to find objections to the view here crudely set forth. At present, however, it is simply a tentative hypothesis, and is placed as such before the Society.

ON INGRAVESCENT APOPLEXY

A Contribution to the Localization of Cerebral Lesions

Medico-Chirurgical Transactions, 1876

SIR THOMAS WATSON, following Abercrombie and corroborating by his own observation the distinctions laid down by Abercrombie, describes *three forms of apoplectic attack.*

"In the *first form* of the attack, the patient falls down suddenly, deprived of sense and motion, and lies like a person in a deep sleep; his face generally flushed, his breathing stertorous, his pulse full and not frequent, sometimes below the natural standard. In some of these convulsions occur; in others rigidity and contraction of the muscles of the limbs, sometimes on one side only.

"Now, respecting persons seen in this condition, the immediate prognosis is uncertain. Some die in a short time and much blood is found extravasated within the cranium. Some die after a rather longer interval, and then we often find serous effusion only. And in some that die early, no effusion either of blood or of serum can be detected. Some recover altogether without any ill effect of the attack remaining. Others recover from the coma, but are left paralytic of one side, and with some imperfection of speech, or of one or more of the senses. And this paralysis and imperfection may disappear in a few days, or gradually depart, or remain for life.

"In the *second form* of attack, the coma is not the earliest symptom. The disease generally begins with sudden and sharp pain in the head. The patient becomes pale, faint, and sick, and usually vomits, and sometimes, but not always, falls down in a state of syncope, with a bloodless and cold skin, and a feeble pulse. This also is occasionally accompanied by some degree of convulsion. Sometimes he does not fall down, the sudden attack of pain being accompanied by slight and transient confusion. In either case he commonly recovers in a short time from these symptoms, and is quite sensible and able to walk; but the headache does not leave him. After a certain interval, which may vary from a few minutes to several hours—and Dr. Abercrombie records cases in which it was even much longer—the patient becomes heavy, forgetful, incoherent,

and sinks into coma, from which he never rises again. In some instances, paralysis of one side occurs; but perhaps more often there is no palsy observed. The disease when it comes on in this way is much more uniform, and of much worse omen, than when it comes after the former fashion."

The *third form* of attack described by Dr. Abercrombie can scarcely be said to be an apoplectic attack at all; indeed he himself includes this form in the class of paralytic cases. It is characterized by sudden loss of power on one side of the body, and frequently by loss or impairment of speech, without loss of consciousness.

To the *second form of attack* the name *Ingravescent Apoplexy* has been given. This I now proceed to consider. Its features are the absence of loss of consciousness at the outset, the gradual accession of symptoms, the almost invariably fatal result, and the large amount of blood found to be extravasated. I ought to add that vomiting invariably occurs at an early period of the case. My observations show, that in these cases not only is the haemorrhage large, but that its situation is within certain limits constant, and I believe that an anatomical explanation can be given of all the phenomena characteristic of the attack.

The seat of the extravasation is on the outer side of the extraventricular corpus striatum (lenticular nucleus) between this ganglion and the external capsule, a plane of fibres which separates it from the convolutions of the island of Reil, and it has appeared to me that the large amount of haemorrhage is explained first by the size of the vessel or vessels ruptured, and secondly by the very slight resistance to extravasation. Also that the absence of loss of consciousness in the early stage of the attack is explained by the fact, that until the quantity of blood poured out is considerable there is no extensive rupture of fibres or ploughing up of grey matter, and probably no compression of brain substance.

Examination of the Brain. A hemisphere in which haemorrhage has occurred in the situation, and to the amount here in question, will present the usual external appearances suggestive of internal pressure, flattening of the convolutions, exclusion of blood from the minute vessels on the ridges, and obliteration of the sulci. If the examination is made simply by the usual sections many interesting points will escape observation. The fissure of Sylvius should be freely opened out, when less moisture will be found in that of the side on which the lesion has occurred than in its fellow, and the pia mater will contain less blood; more remarkable, however, will be the condition of the convolutions of the insula, which will be flattened out and obliterated, so that the island of Reil has the appearance of a smooth oval

elevation. Usually an incision into it at once exposes the clot. Occasionally, indeed, the thin layer of nervous matter over the effused blood is torn in the examination, especially when the clot extends under the anterior perforated space.

I have never seen haemorrhage into the fissure in these cases, though I have known the blood enter and fill the ventricles, burst through the floor of the third ventricle into the interpeduncular space and travel beneath the arachnoid to the Sylvian fissures, finding its way far into the one on the opposite side from the original lesion, but being excluded by pressure from the one near which it was originally effused.

After exploration of the Sylvian fissures the examination may be continued by the usual sections from above downwards. The blood may have penetrated the ventricle or it may have ploughed up the hemisphere on the outer side of the ventricular cavity. Occasionally in the latter case it even reaches the surface of the brain; in the only instance in which I have seen this, the blood had split up the ascending gyri on each side of the sulcus of Rolando (it never, so far as I know, escapes into the bottom of a sulcus). In either case, whether the clot is intra- or extra-ventricular, unless special care be taken it will probably be concluded that the corpus striatum is destroyed, or at least torn by the blood, whereas it will usually be found entire and intact, or only penetrated by a process of the clot, but completely dissected off from the hemisphere proper and displaced inwards.

A detail of some little interest may here be added, namely, that when the blood has entered the lateral ventricle, while all the ventricles may be filled and the cornua distended by clot and fluid, the descending cornu on the side of the lesion will be all but empty, the blood being excluded from it by antecedent pressure on it by the earlier extravasation.

Anatomy of the Hemisphere. It will now be necessary to refer to the anatomy of the part of the brain in which the lesion occurs, and to describe in some detail the relation of parts, the course of fibres, and the distribution of blood-vessels. For this purpose drawings of sections of the brain are shown, which I venture to interpret from my investigations on the course of the fibres of the hemispheres.

The internal capsule, as it has been absurdly called, is constituted by the ascending and diverging fibres which enter the cerebrum below as the crus between the extra-ventricular corpus striatum and the thalamus, and emerge above from the region of the central ganglia to enter the hemisphere proper as the corona radiata. If any fibres of the crus pass uninterruptedly to the

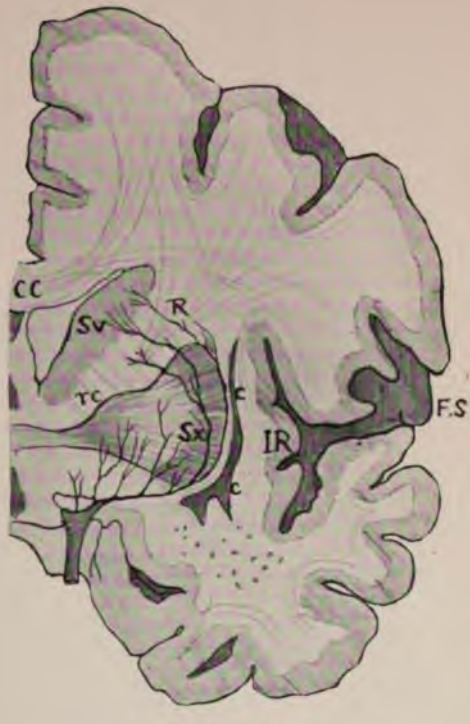

PLATE III.

Transverse section of Brain at Anterior Perforated Space, showing origin of arteries of Corpus Striatum. After Duret. C.C., Corpus Callosum; Sv, Ventricular Corpus Striatum (nucleus caudatus); Sx, Extra-ventricular Corpus Striatum (nucleus lenticularis); rc R, Fibres entering below rc from Crus Cerebri, emerging above R as Corona Radiata, constituting so-called Internal Capsule; FS, Fissure of Sylvius; IR, Island of Reil; c, thin layer of grey matter, Claustrum, between which and Corpus Striatum is thin plane of proper white fibres of Corpus Striatum called External Capsule.

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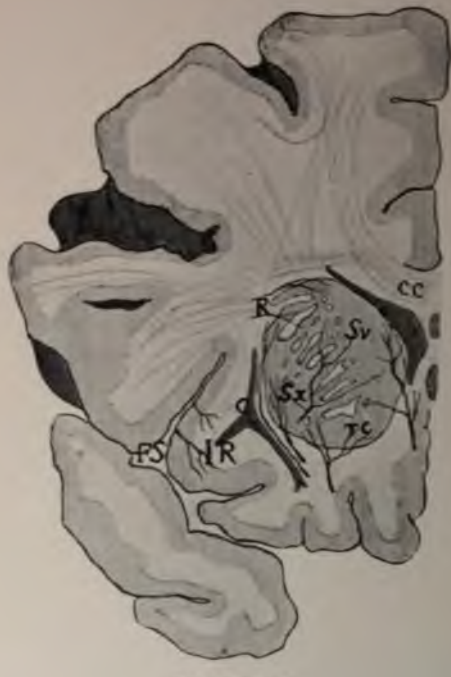

PLATE IV.

Transverse section of Brain in front of Optic Commissure, showing distribution of arteries of Corpus Striatum. After Duret. C.C., Corpus Callosum; Sv, Ventricular Corpus Striatum (nucleus caudatus); Sx, Extra-ventricular Corpus Striatum (nucleus lenticularis); rc R, Fibres entering below rc from Crus Cerebri, emerging above R as Corona Radiata, constituting so-called Internal Capsule; FS, Fissure of Sylvius; IR, Island of Reil; C, thin layer of grey matter, Claustrum, between which and Corpus Striatum is thin plane of proper white fibres of Corpus Striatum called External Capsule.

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hemisphere it is in this tract. As the crus passes into the interval between the ganglia its two divisions, presumably motor and sensory, the crusta and tegmentum, are distinct one from the other, but this separation is soon lost, and in the corona radiata there is a complete intermingling of fibres, so that no distinction can be made between those which in origin and function are different.

We are not here concerned with the rearrangement of fibres which takes places as the crus becomes corona radiata, but the increase in number is obviously very great. To this increase the thalamus contributes conspicuously, the corpus striatum less conspicuously, though probably more abundantly. It is not easy to trace fibres arising in the intra-ventricular corpus striatum and proceeding onwards to the hemispheres, but the radiating fibres in passing between the head of this ganglion and the extra-ventricular division of the corpus striatum separate into bundles between which the soft grey matter of the two portions dips in and comes into communication, probably giving off fibres. Between these bundles also M. Duret has followed to the intra-ventricular corpus striatum arteries which enter at the anterior perforated space, and far more numerous and larger veins which pass from the extra-ventricular corpus striatum and the hemisphere towards the ventricle, to appear there as the venae Galeni.

The external capsule will also require a detailed description. It is seen in the various sections as a thin layer of white fibres separating the grey substance of the corpus striatum externum from the cortex of the insula, and it includes a plane of grey matter, the claustrum, which appears in the drawings as a thin streak. The convolutions of the island of Reil lying upon the outer wall of the corpus striatum have absolutely no connexion with this ganglion, and the claustrum and all outside of it can be removed without disturbing a cell of the ganglion or a fibre arising in it. The corpus striatum then has only a very thin lamina of fibres of its own, forming its proper capsule externally; an equally thin lamina of fibres arising in its cells encases it on its inferior aspect. About these further particulars must be given.

When by careful dissection the proper wall or capsule of the extra-ventricular corpus striatum has been exposed, the temporo-sphenoidal lobe will almost have disappeared and the fissure of Sylvius will be converted into a wide hollow or groove curving round the ganglion which, as the brain lies base upwards, has the appearance of a smooth elevation embracing the crus. The fibres of distribution forming this capsule radiate forwards, backwards and outwards in all directions from two patches of the grey substance of the ganglion which have become exposed at the summit,

one beneath the uncinate lobule, the other just at the outer side of this point. Those passing outwards, forming the proper "external capsule," instead of crossing the Sylvian hollow, as would be expected from the apparent continuity of fibres across it, to the remains of the marginal convolutions of the fissure, dip in between other larger masses of fibres which are those of the corona radiata emerging from the "internal capsule," so that a crossing or intersection here takes place almost at right angles.

A more important anatomical fact relating to this plane of fibres forming the external capsule is that, as Duret has shown, the large arteries which enter the hemisphere at the anterior perforated space for the nutrition of the corpora striata pass upwards between the capsule and grey matter. To this further reference will be made in the concluding remarks.

CASE 1.—*Hemiplegia of left side. Lateral deviation of head and eyes. No loss of consciousness. Sensation greatly impaired. Vomiting. Sleep or stupor. Seven or eight hours after the attack, sudden access of stertor and profound coma. Death in twenty minutes. —Haemorrhage between the grey substance of the corpus striatum externum and the plane of fibres separating it from convolutions of insula cutting off hemisphere from central ganglia, and ultimately bursting into lateral ventricle. Pulmonary, splenic and hepatic haemorrhages.*

This case, though not the first of the kind observed, is placed first because I was able to watch it from an early period of the attack, and to recognize the seat of the lesion.

W. S., aged 50, foreman of some lime-works in the immediate neighbourhood of St. Mary's Hospital, was admitted into the hospital about 2.15 p.m. on February 9, 1871, and at once seen by me.

He was an intelligent, steady, sober man, had never had any serious illness, but had lately suffered occasionally from headache, and his appetite had been variable. He had just gone to work after dinner, when he felt giddy and sat down. He appeared to be confused, but never lost consciousness. Within a few minutes he was seen by a friend to whom he said he felt giddy, but should soon be better; his friend, however, noticed that his head seemed to be turned to the right, that the left arm hung powerless, and that he did not appear to know he had a left arm. He was at once brought to the hospital, and, being at hand, I was called to him.

He answered questions, gave an accurate account of the attack, and expressed anxiety about some money which was in his purse, but his manner was excited and he called his friend by name and gave him directions, though he was no longer present. He tried

also to take hold of the paper on which the notes were being made, and resisted when his hand was restrained.

The face was not perceptibly distorted, scarcely so even when he spoke, and he could whistle though not so well as usual. The head was turned so that the face looked over the right shoulder, and the eyes were carried to the extreme right of the palpebral fissures, the deviation of head and eyes being most marked. He was unable to look straight before him, still less to the left. Left arm and leg quite powerless and relaxed; there was slight reflex action when the sole was tickled. Sensation was greatly diminished in face, body, arm, and leg of this side.

Face slightly flushed; arcus senilis present; pupils natural. Pulse 108, rather long and firm, but not strong. Heart-sounds normal; no reduplication; no evidence of hypertrophy. Respiration natural.

A tendency to sleep gradually manifested itself, and having by careful observation come to the conclusion that the case was one of those in which a fatal termination is almost certain and in which a large clot is invariably found I determined to bleed. My resident medical officer, Mr. Boone, opened in succession three veins, two in one arm and one in the other, but was unable to obtain more than a very trifling flow of blood. That the veins were really opened was shown by passing a probe along them upwards and downwards. I then proposed to bleed from the jugular, but it was impossible to bring about the necessary fullness of the vein. While considering the desirability of dividing the temporal artery the condition of the patient underwent a change, the face became bedewed with perspiration, the pulse was softer, and very shortly he vomited violently, bringing up large quantities of undigested food, and during the effort urine and faeces were passed involuntarily. He repeatedly provoked fresh acts of vomiting by putting his fingers into the throat in spite of remonstrances. This was about 2.45 p.m.

I did not think it desirable to persist in the attempt to bleed, and a dose of calomel and castor oil was ordered, cold being applied to the head. The patient continued to sleep during the afternoon, but could be roused and when awake was quite sensible; he took a little milk and vomited occasionally.

At 10 p.m. stertor suddenly set in, and he was found to be quite unconscious. The stertor continued though he was turned on his side, as recommended by Dr. Bowles (a proceeding which is usually perfectly successful in eliminating the element of danger which stertor adds in these cases), and the respiration was catching. The heart was acting violently, at the rate of 150 beats per minute.

During this final attack the lateral deviation of the head and eyes ceased. This was observed for fifteen minutes. The right pupil was large, the left small. At length, twenty minutes after the stertor set in, the breathing suddenly stopped, but the heart continued to beat and the pulse could be felt for seven and a half minutes afterwards, becoming gradually more feeble. At the moment of death the left pupil dilated.

Post-mortem examination.—Brain—a little flattening of convolutions of right hemisphere; a superficial, slight, meningeal haemorrhage of the anterior extremity of frontal lobe on this side. Blood seen to have issued from fourth ventricle, between medulla and cerebellum. Atheromatous patches in arteries generally, with dilatations. When fissure of Sylvius opened out, convolutions of island of Reil on right side seen to be flattened out; the entire insula being large and soft.

When hemispheres sliced nothing abnormal met with above level of corpus callosum. When lateral ventricles opened, blood-stained fluid and a small clot found in left; a rupture in septum lucidum; blood in third ventricle, iter, and fourth ventricle, and in all the cornua of both lateral ventricles. In right lateral ventricle a large black clot, and this being removed a fissure seen (to the outer side of the corpus striatum and thalamus) extending along the entire length of the ventricle, crossed by venae Galeni and processes of the ependyma left untorn. From this fissure, which was wide and gaping, black clot protruded, on removing which a cavity was found, two and a half or three inches long, which extended along the entire length of the two central ganglia on their outer side. The outer wall of this cavity was for the most part formed by the plane of white fibres which, arising from the grey matter of the corpus striatum externum, separates this grey matter from the convolutions of the insula (external capsule). The inner wall was formed mainly by the grey substance of the extra-ventricular corpus striatum, which was not ploughed up or penetrated by the blood or softened, but was left entire, and was simply dissected off from the fibres. The lower edge ran parallel to the descending cornu of the ventricle along its whole length, and was close to it; superiorly the rupture into the ventricle had taken place by tearing of the fibres of the corona radiata issuing from the central ganglia (internal capsule).

The haemorrhage had taken place from vessels running from the artery in the Sylvian fissure to the ganglionic substance of the corpus striatum, between the grey matter and the plane of fibres arising from it to pass to the hemisphere. The blood had followed the line of cleavage, and after separating the fibrous capsule from

the grey substance had torn through the corona radiata and burst into the ventricle.

Heart not large; valves fairly healthy.

Lungs.—Extensive haemorrhage into both, the blood black and coagulated; the extravasations had neither the appearance nor distribution of ordinary pulmonary apoplexy.

Considerable haemorrhage had taken place into substance of spleen and small scattered haemorrhages were found in the liver. Kidneys healthy. I am of opinion that the pulmonary, splenic, and hepatic haemorrhages took place during the act of dying, and were due to the mode of death, by suffocation; that, in fact, the extravasation was caused by the prolonged and violent action of the heart after the suspension of respiration.

(Only the abstract of the other cases will be given here, though they were originally stated in full.—Ed.)

Case 2.—*Hemiplegia, left. Sudden in access. Frequent vomiting during next twelve hours. Sopor for three days. Sensation greatly diminished. Rapid formation of bedsores. Death in three and a half weeks.—Extensive haemorrhage between extra-ventricular corpus striatum and plane of white fibres separating it from insula. Mitral disease. Contracted granular kidneys.*

Case 3.—*Apparent drunkenness. Left hemiplegia coming on gradually with hemianaesthesia. Later unconsciousness.—Haemorrhage between corpus striatum and external capsule penetrating into lateral ventricle.*

Case 4.—*Giddiness and staggering. Left hemiplegia; ingravescent in access. Loss of consciousness. Vomiting.—Haemorrhage between corpus striatum and convolutions of insula.*

Case 5.—*Hemiplegia with marked hemianaesthesia. Ingravescent mode of attack.—Haemorrhage between corpus striatum and thalamus and external capsule at posterior part of insula, with penetration of blood through thalamus into ventricle.*

Commentary.—The cases just related furnish sufficient evidence of the association of the ingravescent form of apoplectic attack with haemorrhage of large amount between the lenticular nucleus and the external capsule. The occurrence of extensive extravasation of blood in this situation has already been noted by Charcot, Bouchard, and Duret, and explained by the *large size of the arteries* here found. It is well known that the grey substance of the nerve-centres is much more freely supplied with blood than the white fibres, and that large vessels pass from the middle cerebral artery into the foramina of the anterior perforated space to the grey mass of the corpus striatum. Duret has shown by sections of injected brains that these arteries do not at once plunge into the grey substance

of the ganglion and ramify in it, but run, some of them, one of large size in particular, round nearly its whole external periphery, between the grey matter and the external capsule —a fact which I had observed independently in the course of my dissections. Duret has also shown that they end in tufts and do not give off many branches like other arteries. It is obvious, as pointed out by this observer, that in the large size of the vessels and in their mode of termination, we have an explanation of the liability to rupture and of the profuse haemorrhage.

A further reason not previously taken into account why extravasation of blood in this situation is liable to be extensive is the *absence of resistance*. I have found that the plane of fibres forming the inner surface of the external capsule, though the fibres no doubt arise from the cell processes of the corpus striatum, is detached from the grey substance with extraordinary facility, so that the blood only performs a natural dissection in following the line of cleavage between them. In thus separating the capsule from the ganglion it will, moreover, probably cause rupture of other arteries running here.

Not only is there an absence of the obstacle to extravasation which would be offered by interlacing fibres, but the proximity of the fissure of Sylvius and of the descending cornu of the lateral ventricle diminishes the resistance to displacement and consequent pressure which solid brain would oppose. The Sylvian fissure is one of the provisions found in the brain for meeting variations in the vascular turgescence of the cerebral substance by corresponding variations in amount of meningeal fluid, and the room it affords for expansion of the island of Reil can only be fully appreciated when the gyri operti of the latter are seen, as they are in these cases, not merely flattened but completely obliterated, so that the insula appears as a smooth, unconvoluted eminence. Further space is yielded by the compression of the descending cornu of the lateral venticle, and by the displacement of the corpus striatum inwards.

The ready mobility of the parts surrounding the seat of haemorrhage, besides facilitating the extravasation, will also prevent any general or local pressure on brain substance till the mobility is exhausted, and to this I am disposed to attribute the late and *gradual oncome of coma*. The relation between pressure and coma, it is true, is a question still in dispute, and it cannot here be adequately discussed. It is, however, generally admitted that the brain will bear without symptoms the gradual application of pressure, which, applied suddenly, would give rise to coma, and that sudden injury

produces the unconsciousness of shock, or, as it was named by Trousseau, "cerebral surprise," and it is clear that sudden pressure and forcible laceration are avoided in consequence of the anatomical fact here pointed out.

Course of the Extravasated Blood.—The extravasation is, however, rarely confined to the space between the extra-ventricular corpus striatum and its external capsule. When this is the case, the hemiplegia will probably be slight and temporary, and a minute account of the symptoms would be of extreme interest and value. It will easily be understood how the effusion of a considerable amount of blood before resistance is encountered favours the further progress of the extravasation; since, according to the laws of fluid pressure, the distending force is the same at every point of the surface of the blood poured out as at the orifice of the bleeding vessel, and is therefore multiplied indefinitely. It will thus readily make its way, but always in the direction of least resistance; and here, as in all cases of cerebral haemorrhage, the further course of the blood will be determined by the arrangement and relations of the fibres.

When the ruptured vessel is at the anterior part of the corpus striatum, the blood travels forwards, and may penetrate into the frontal lobe; when on the lateral aspect, the blood is directed upwards. The haemorrhage never breaks through into the fissure of Sylvius, nor does the blood ever, so far as I know, enter and plough up the temporo-sphenoidal lobe; to do this it would have to penetrate continuous planes of fibres, which in the absence of antecedent softening never happens. At the upper angle of the extra-ventricular corpus striatum the plane of fibres which forms its proper external capsule, passing upwards, meets the mass of fibres of the corona radiata proceeding outwards, and an interlacement nearly at right angles takes place between them. At the line of intersection, too, the few arteries which are sent from the middle cerebral to the intra-ventricular corpus striatum and to the centrum ovale of the hemisphere pass upwards and inwards, and also the far more numerous and larger veins conveying blood from the extra-ventricular corpus striatum and the mass of the hemisphere to the venae Galeni.

It might have been supposed that the intersection of fibres would oppose a barrier to the further extension of the haemorrhage, but apparently interlacing fibres offer less resistance to rupture than a smooth continuous plane. No doubt, also, the vessels with the perivascular spaces afford a guide and open a way to the blood, since it almost invariably happens that the corona radiata is torn through partially or completely along the

line of its emergence from the region of the central ganglia. The extravasation then either reaches the ventricle or ploughs up the hemisphere; in the latter case it may be directed into a longitudinal course in the substance of the parieto-frontal lobes by the mass of fibres of the great longitudinal commissural system, or may follow the central radiating fibres into the ascending parietal convolutions, which it splits up in the direction of their length.

It is the ingravescent mode of attack which constitutes the distinguishing character of the class of cases I have endeavoured to describe, and which I associate with haemorrhage between the corpus striatum and external capsule. With the progress of the haemorrhage new symptoms arise, which will differ according to the course taken by the blood. So far as the cases here related throw light on the symptoms attending different degrees and direction of further structural damage, it would appear that in all there were hemiplegia, hemianaesthesia, vomiting and sopor. A noteworthy point is the very slight degree of facial hemiplegia observed, attributable perhaps to the distance of the lesion from the first or internal part of the lenticular corpus striatum, which is said to be specially concerned in the movements of the face and eyes. At the same time lateral deviation of the eyes was nearly always present.

The patient in Case 2 survived, for three weeks, complete separation of the extra-ventricular corpus striatum and thalamus from the hemisphere. The rapid formation of bedsores indicated great impairment of the nutritive functions in the paralysed parts. In all the other cases the blood reached the ventricles, and a fatal termination occurred in a few days. In Case 1 laceration of brain fibres was attended with the symptoms enumerated and marked conjugate deviation of the head and eyes, while rupture into the ventricle caused profound coma and speedy death. In Cases 3 and 4 extensive ploughing up of the hemispheres with penetration into the ventricle at an indeterminate period gave rise to early rigidity of the paralysed parts, and constant movements of the limbs not paralysed. There was gradual loss of consciousness in both, but less profound and less durable in the latter, in which the blood took a direction forwards. In Case 5 the lesion was further back, and the injury to the thalamus greater than in any other case, and the impairment of sensation was very decided, while the loss of consciousness was not for some time so profound. The size of the clot was, however, much smaller.

It will be noticed that *loss of sensation* to a certain and indeed considerable degree has accompanied the hemiplegia, and this not only when the thalamus has been damaged, or when the posterior part of the internal capsule so called has been involved in the

lesion, but when the haemorrhage has been too far forwards for either occurrence. To this I beg to call special attention as hemianaesthesia has recently been too exclusively referred to lesion of the posterior part of the internal capsule. There can be no doubt that injury of the fibres at this part will cause hemianaesthesia as observed by Charcot, Bournville, and others, and shown experimentally by Carville and Duret and others. I desire simply to point out that impairment of sensation can be induced by lesion elsewhere, and especially in the situation I have spoken of outside the central ganglia and beyond the internal capsule.

This does not constitute a valid objection against the sensory function of the thalamus. The impairment of sensation is caused in the same way as the motor paralysis, which in these cases is due not to destruction of the corpus striatum, or to a lesion cutting it off from communication with the medulla and cord, but to separation of this ganglion from the hemisphere by rupture of the fibres of the corona radiata, and the same lesion which divides the communication of the corpus striatum with the hemisphere divides that of the thalamus. The fibres of the thalamus do not go merely or even mainly to the posterior part of the hemisphere, but pass forwards and outwards beneath the intra-ventricular corpus striatum to emerge from the region of the central ganglia in the corona radiata of which they everywhere form an important part. They are thus implicated when the corona is torn, and the cases show that separation of the thalamus from the hemisphere is attended with impairment of sensation, as well as damage to the ganglion, or its separation from the sensory tract of the cord.

LETTSOMIAN LECTURES ON SYPHILITIC AFFECTIONS OF THE NERVOUS SYSTEM (*Abbreviated*)

The Lancet, 1874, Vol. I

LECTURE I

Syphilis as a Cause of Disease in the Nervous System

Stages and Forms of Syphilis in which the Nervous System is implicated—Pathology—Syphilitic Affections of Nerves—Neuralgias and Local Paralyses

Gentlemen,—The subject I propose to bring before you in these lectures is, as you know, the Syphilitic Affections of the Nervous System. There are, perhaps, other topics on which I might have spoken with greater authority, and there are certainly other physicians more competent than myself to treat of the one I have chosen. My justification before you is, that, while numerous interesting and valuable observations have been placed on record in various periodicals and reports, the subject has not yet been treated of as a whole by any English physician, and the profession generally has not yet become aware of the frequency of syphilitic affections of the nervous system, and in consequence of this a class of diseases, painful and fatal if not recognized, but easily curable, and which indeed furnishes us with some of our most striking instances of success, frequently escapes notice and treatment. An additional justification which I make of myself is the interest I have long taken in nervous physiology and pathology, and the opportunities which syphilis has afforded me for the investigation of the effects of morbid changes in the nervous centres.

It will be accepted as a sufficient evidence of the frequency of syphilitic disease of the nervous system that I shall in these lectures have little occasion to quote the observations of other physicians, but that I shall be able abundantly to illustrate almost every variety of syphilitic affection from my own experience, obtained entirely at a general hospital or in private practice, and without the special field of observation afforded either by an institution for venereal diseases or by a hospital for diseases of the nervous system.

The Stages of Syphilis.—In considering the diseases of the nervous system connected with syphilis, the first question which arises is, At what period of syphilis are these affections liable to be introduced? Syphilis is considered to exhibit primary, secondary, and tertiary stages, or we speak of primary, secondary or tertiary manifestations. These designations have relation, not to mere lapse of time, and not always even to order of appearance, but more strictly to order of lesion than to order of succession. We see, in one case, many years after the infection, an intractable scaly eruption of the skin, or obstinate ulceration of the tongue or the commissure of the lips, recurring again and again, and following, perhaps, two or three distinct sets of symptoms. These are secondary in character, or intermediate, as Mr. Hutchinson would call them, notwithstanding the interval of time and the successive forms of manifestation.

In another case we have a woman impregnated by a syphilitic husband, and infected through the foetus. In her the first symptom may be a destructive pharyngeal ulcer, or a node; or, in another case, primary infection may be followed at once by a pustulo-crustaceous eruption, and this by periostitis, rupia, and other manifestations of a like kind. We call these tertiary, or, at least, we must treat them as such. Again we have the perplexing instances of hereditary syphilis. A child of 10 or 12 years is found with an ulcerated gumma of the tongue, or ulcer of the palate. Or we find a healthy young woman, of known character and virtue, with cutaneous gummata and tubercles, though the first symptoms belong strictly to the class of tertiaries.

When, therefore, we are endeavouring to determine the stage of syphilis to which a given case belongs, time is not the main element in the question, but the associated or antecedent symptoms, and the distinctions which we shall find it important to bear in mind are those pertaining to the character of the lesion. If we admit into our catalogue of nervous affections all the symptoms arising in the course of syphilis which are capable of being referred to the nervous system—the wandering rheumatoid pains felt in the muscular structures in early syphilis, the osteocopic symptoms of later stages, sleeplessness, irritability, change of disposition—the liability is co-extensive with the disease.

We all recognize the distinction between the disorders of the nervous centres due to the circulation in them of poisoned blood, and the diseases of those centres produced perhaps by some blood poisons; as, for instance, between the common delirium of enteric fever occasioned by a heated and impure state of the blood in the disease, and the exceptional meningitis set up

by the same state. There is the same distinction in syphilis between the disorders of the nervous function and the morbid conditions of the nervous structures which it can induce. With this limitation, which will greatly economize my time, and permit me to devote more attention to the more important part of my subject, we shall find that *the nervous affections may arise either in the secondary or in the tertiary stage of the disease, but far more frequently in the latter.*

The affections of the two periods, moreover, are not identical, and I think the diversity will be found more considerable than has generally been supposed. The difference has been recognized by most observers, and it is what might have been anticipated from the different clinical characters and different pathological tendencies of syphilis in its various stages. It is even better understood, as it appears to me, by reference to the theory of Mr. Jonathan Hutchinson, which brings syphilis into the class of continued eruptive fevers. According to this theory the secondary stage of syphilis represents the fever—the tertiary stage so-called, the effects produced upon the solids and liquids of the organism by the febrile process. The tertiary stage becomes thus, not a part of the disease itself, but a consequence of it, corresponding to the sequelæ of fever, such as dropsy in scarlatina and scrofulous affections after measles.

Whatever view may be taken of tertiary manifestations, whether they are to be considered as the continuation or as the consequence of syphilis, the truth of the analogy between syphilis and continued fever appears to me undoubted. They have in common a period of incubation and a febrile stage which runs a more or less definite course. In this febrile stage, which is attended with symmetrical cutaneous manifestations and disseminated lesions in the internal organs, the poison is reproduced in the system, and the individual who is the subject of the disease becomes a source of contagion. Finally, one attack usually confers future immunity from the disease.

Now, just as in fevers we may have pneumonia or meningitis not distinguishable by any anatomical characters from pneumonia or meningitis due to other causes, and recognized clinically by the supervention of these conditions upon those of the fever, so, in the secondary stage of syphilis, there may occur spinal or cerebral congestions or inflammations which have no peculiarity to indicate the syphilitic character of the affection, and this has to be ascertained almost entirely from previous history or from existing manifestations of syphilis. In tertiary syphilis, on the other hand, the morbid processes set up are altogether peculiar, as will be described, and the symptoms are often sufficient of themselves to

establish the nature of the case in the absence of collateral evidence. As they will engage our attention later, when considering the affections of different parts of the nervous system, I shall not dwell longer upon them now, but proceed to the consideration of another question—whether, namely, there is any particular form of lesion, or any particular course of the subsequent constitutional manifestations, which is attended with special liability to affections of the nervous system.

Type of Syphilis leading to Nervous Affections.—An answer to this question is scarcely to be obtained directly. The surgeons who have seen and treated the primary sore or the early constitutional symptoms are not often consulted for epilepsy or paralysis, which, in the mind of the patient, are never for a moment associated with the former affection, appearing as they do after an interval of time, perhaps, which has allowed even the occurrence of the disease to be forgotten, or has reduced it, in his recollection, to an accident of trifling significance. The family medical man, or the physician, on the other hand, called to deal with the nervous affection, has not watched the course of the disease in the earlier stages. Indeed, he may have been expressly kept in ignorance of it.

From the cases which have come before me, and from what I have seen of syphilis affecting other organs, confirmed by inquiries which I have made of surgeons who have extensive opportunities of observing the disease in all its stages, I have formed an opinion that it is chiefly in persons in whom *the secondary affections have been transient* and insignificant or even absent, or in those in whom the tertiaries arrive early or primarily, that the nervous system is liable to suffer. I am corroborated in this view by the statements of Gross, Lanceraux, Buzzard, Moxon, and other writers, and it is scarcely possible otherwise to explain *the entire absence of syphilitic history in many cases obviously of a syphilitic character.*

But this is exactly what we see in the case of other tertiary manifestations. We are almost daily called upon to make a diagnosis of syphilis in the absence of a history, and in the face of positive assertions that the patient has never been subjected to any syphilitic infection, or suffered from any specific manifestations. In the case of women, and especially of wives, this is not to be wondered at. They are often absolutely ignorant that they have contracted the disease. For my own part I have long come to the conclusion that it is useless and cruel to ask a woman if she has had syphilis. The primary lesion, especially situated on internal parts, as on the cervix uteri, may give rise to so little inconvenience, and any discharge from it is

so readily confounded with other secretions, that it may pass entirely unperceived. In women, again, the disease is often communicated by a syphilitic foetus. The father is a subject of syphilis, but has no lesion secreting poison to inoculate directly the mother; the ovum, however, is affected by the father's disease, and through the interchange between the foetal and the maternal blood the mother becomes contaminated. In these cases the secondary stage of the disease is frequently altogether absent as well as the usual primary or initial lesion, and tertiary manifestations are developed forthwith.

Both sexes, again, may be the subject of inherited syphilis, which is often betrayed by traces or histories of infantile or early manifestations, or by the teeth, the nose, the conformation of the head, for an account of which medicine is for ever indebted to Mr. Jonathan Hutchinson. These valuable signs, however, may be absent, and, as I have already said, we confidently pronounce many affections to be syphilitic simply from the special character of the manifestations. As syphilitic affections are frequently curable, the patients should have the benefit of the doubt. This is so far recognized that it has become a sort of rule among those who have given attention to the subject, when in doubt, to give iodide of potassium.

The fact that in a large proportion of the cases in which the nervous system is affected with disease arising out of syphilis, the secondary stage has been brief or imperfectly represented or absent, and that very rarely is the history of syphilis prominent, renders it very important to make out, if this be possible, the distinctive character of syphilitic affections of the nervous system which of itself will serve to determine the diagnosis. We shall see later how far this is attainable, and one intermediate step towards the elucidation of the problem is the consideration of the pathology of the syphilitic changes occurring in nervous structures.

Pathology.—The symptoms to which any disease of the nervous system gives rise will result primarily from derangement of function of the part affected, but the kind and degree of such disturbance will be greatly influenced by the nature of the morbid process and by its rate of progress. It will make a difference, for example, whether a tumour at a given situation in the brain or cord has its starting point on the surface or in the substance, whether it increase rapidly or slowly, whether it grows at the expense of the nervous structure, causing no actual increase of volume, or adds to the contents of the cavities, and displaces or compresses the nervous matter. In considering, therefore, the morbid conditions to which syphilis gives rise, we have to take into account

not only the morbid anatomy but the general habit of the disease.

There are very large materials from which I might draw the account to be placed before you of morbid changes produced by syphilis in the nervous structures. I shall avail myself almost exclusively of those of Drs. Wilks and Moxon, whose contributions are at once ample, clear and comprehensive; indeed I know nothing more admirable than their descriptions of syphilitic disease in the Guy's Hospital Reports. According to Dr. Wilks, in syphilis there is a disposition to the effusion of a low form of fibroplastic material in nearly every tissue of the body. When the exudation comes to be examined after death, it has generally had a long existence in the organ in which it is found, and it presents under the microscope fibroplastic elements, small nuclei, fatty granules, and some amorphous matter. As a rule the deposits are hard and fibrous, and not soft, as is suggested by the term gummatous usually applied to them, but they may undergo secondary softening.

There is nothing specific in the individual elements, and, indeed, the notion of a specific structural element in any disease is now generally abandoned; but characteristic peculiarities exist which have been more exactly described by Dr. Moxon. He says: "If one looks over a series of syphilitic changes and compares them with any other form of changes, one finds that the syphilitic cases have characters by which they are practically easily distinguished: (1) Generally a small part of the organ is attacked and the remainder is left quite free. The disease is strictly localized in the spot it affects. (2) Its outer part is composed of fibrous tissue, which can be seen to represent the natural fibrous supporting elements of the part in a state of augmentation, while the functioning elements of the part have dwindled away. It is a local sclerosis. (3) Its central part shows the now celebrated caseous or gummatous faint yellowish matter of more and more elastic consistence and less and less friability and curdiness, generally rather sharply distinguished from the fibrous outer part, and sometimes softening down or calcifying. (4) There are signs of more acute inflammation in the immediate neighbourhood, showing lymph, etc., or adhesions to the parts around. (3) and (4) may be absent. Such patches, sharply contrasting with more healthy tissue immediately about them, and (5) distributed more or less widely in a variety of organs, but especially in the testes and liver, are not a general thing which could be passed over as a common accident. Their characters attract attention. A syphilitic gumma in muscle or brain is so unlike anything else that, if seen for the first time by one who knows the rest of the common run of

pathological changes, it demands from him some recognition of its peculiarities. In short, it is not common, but specific in the strict sense of the word. . . .

"Syphilis attacks the surface of the brain and its membranes, it attacks them in limited spots, and it spreads slowly. The morbid changes are, on the one hand, adhesions of the membranes to each other and to the surface of the brain by means of an adventitious material of firm consistence and yellow colour, which may be called lymph, but is harder, tougher, and more opaque. This exudation may be found at any part of the surface; it invades and destroys the grey matter, interferes with the supply of blood, and, when it occupies the membranes at the base of the brain, surrounds and involves the nerves in the intra-cranial part of their course."

On the other hand, the syphilitic deposits may take the form of a distinct tumour of fleshy aspect, vascular externally, but presenting at the centre the well-known gummatous character. Or the deposit may be small and circumscribed, but multiple, firm, and hard as to consistence, and yellow in colour. Around the foreign bodies may be more or less inflammation or softening in the spinal cord. While the general characters are similar, there are differences in detail, which will be described later.

But all these deposits of lymph, whether diffused or circumscribed, are met with in the tertiary stage of syphilis. The differences in the clinical history of secondary and tertiary syphilis, and in the treatment required by these two stages of the disease, are such that nothing short of the overwhelming evidence we possess would convince us that they are only two periods of the same affection; and we may well look for differences in morbid anatomy. The differences found are, moreover, such as would be explained by considering secondary syphilis as fever, of which the lesions of tertiary syphilis are the sequelae. Looking upon inflammation as the result of the disturbance of the relations between the blood and the tissues, in the one we have this relation disturbed by a morbid condition of the blood, in the other by a deteriorated state of the tissue elements.

In *secondary syphilis* the pathological conditions are chiefly evidence of meningitis, old or recent, or of congestion, and very frequently no appreciable lesions have been discovered, the same conditions, in fact, as produced by other blood poisons. In *tertiary syphilis* are found the peculiar localized changes described. In addition to the morbid changes in the nervous structure proper and in the vascular meninges, the brain or spinal cord may be invaded by gummatous tumours springing from the dura

mater or the bones, or may be affected by extensive inflammation from carious bone. Or again the blood supply may be cut off by obstruction of an artery from syphilitic disease of its walls.

Scheme of the Lectures.—The scheme I have laid down for myself in these lectures is to describe successively the effects of syphilitic disease on the nerves, on the spinal cord, and on the different parts of the encephalon. I am not aware that this has as yet been systematically attempted, but, though it would have been easier, and perhaps safer for me to have followed the beaten track, and speak of symptoms or groups of symptoms; of affections of sensation, motion or intelligence; of painful, convulsive, and paralytic affections; of affections of the nervous system without referring them to any definite seat and form of lesion, I have preferred to try to make use of our increasing powers of localizing and defining the morbid conditions from the symptoms to which they give rise, and to make the organic changes the basis of my classification.

I come first, then, to the syphilitic affections of the nerves, which will comprise the neuralgias and the local paralyses.

Neuralgia.—Of the pure neuralgias I have little to say; I do not think they are very common; they do not differ materially from the ordinary form of neuralgia. The cervico-occipital, the cervico-brachial, the sciatic, and some of the visceral nerves, have been found to be affected much in the same proportion as when no specific influence is in operation; and Dr. Anstie has given a clue to the occurrence of neuralgia in syphilis, which is that the subjects are constitutionally neurotic, and syphilis is one among the many causes which may bring out the tendency. It is more common in the secondary than in the tertiary stage, and may even precede the first cutaneous eruption. If recognized, the most successful treatment will be that appropriate for the stage of syphilis in which it occurs. Sometimes the treatment of neuralgia as such will succeed, as we sometimes see in other cases in which neuralgia is suppressed, in spite of the continued existence of an exciting cause, by operating against the predisposing condition of the system. A form of nerve pain, differing from the ordinary neuralgia, more dull, aching and continuous, may be caused in the tertiary stage by neuromata (very rare), by pressure from a node or gummatous tumour, or by conditions giving rise to sensory and motor paralysis, which I shall now proceed to consider.

Affections of Nerves.—We may have local paralysis or syphilitic affections of every cranial or spinal nerve. With the exception of a transient loss of power sometimes seen in the ocular muscles, paralysis of individual nerves is almost invariably a tertiary phenomenon. It may be produced in various ways, by a neuroma, by

the inclusion of the nerve in a gummatous tumour—a very rare occurrence of which I have seen a few distinct specimens—but usually the question of causation lies between periostitis about the orifice of exit of the nerve from the cranium or spinal canal, and meningeal exudation involving the nerve during the intra-cranial or intra-spinal part of its course. The latter is by far the most common cause, as will be seen from a consideration of the cases.

Any of the cranial nerves may be paralysed from the consequences of syphilis, but some much more frequently than others. I shall not here speak of the affections of the special senses—loss of the sense of smell, blindness or deafness. To speak only of vision: it may be impaired or lost in consequence of syphilitic inflammation of the choroid coat or retina, or from double optic neuritis consequent upon a syphilitic tumour in any part of the brain, or from pressure upon the optic tract. Deafness, again, which is connected with syphilis, may be due to affections of the auditory mechanism or the auditory nerve.

Leaving these out of the question, then, the cranial nerve most frequently affected is the third. Sometimes the only result is mydriasis, or loss of accommodation, or dilatation of the pupil, or ptosis of the eyelid: but more commonly the entire nerve suffers, and in addition to the ptosis and dilatation of the pupil, we have external strabismus and immobility of the globe of the eye. The twofold fact that the third nerve is the most frequently affected and often the only nerve paralysed, and, again, that portions of the nerve may suffer before the others, is explained by the habit of locality of syphilitic exudation, of which the interpeduncular space at the base of the brain traversed by the third nerve on its way to the cavernous sinus is the favourite seat, and it is conclusive evidence that paralysis is not due to periostitis at the orifice of exit. The nerve also has been found compressed by a gummatous tumour of the sella turcica of the sphenoid bone, and I shall give an illustration as an example in which both nerves were affected.

Paralysis of the sixth, the evidence of which is internal strabismus, is, perhaps, next in frequency. Paralysis of the fourth, the remaining motor nerve of the eye, is not common; it is shown by double vision without an obvious squint, the two images being obliquely placed with respect to each other, and receding when the patient looks down, since this brings the superior oblique into action, and approximating each other, and finally equalized as the eyes are raised, so that vision is single when the patient looks up.

Paralysis of the seventh, unlike oculo-motor paralysis, is more frequently caused by pathological conditions which are not of syphilitic origin. The features which it presents, the total paralysis

of the side of the face with inability to shut the eye, are too familiar to need discussion. Paralysis of the fifth may be seen in loss of sensation in the face and paralysis of the masseter; of the glosso-pharyngeal in difficulty of swallowing; of the hypoglossal in paralysis of one side of the tongue and the sterno-hyoid and thyroid muscles; and of the spinal accessory in the larynx and palate, and sterno-mastoid and trapezius muscles.

I will conclude with a few *cases of paralysis of individual nerves*, first cranial, then cervico-brachial, and then of the lower extremities. The first is a case of paralysis of the left third nerve, slight right hemiplegia, and ultimately paralysis of the right as well as the left third. . . . My interpretation of his symptoms was that syphilitic inflammation with gummatous deposit had invaded the interpeduncular space, involving the two third nerves, and at one time threatening to invade the left crus. It is possible that there might have been here, as in a similar case that I have seen, a gummatous tumour of the sella turcica.

The next case is one of paralysis of the first and second divisions of the right fifth nerve, the fourth nerve, and the palpebral division of the third nerve on the same side. . . . No single lesion would account satisfactorily for the paralysis of the different nerves involved. The superior maxillary division of the fifth has but a very short independent course within the cranium from the Gasserian ganglion to the foramen rotundum, and it may be concluded that a lesion involving the two divisions of the fifth was near the apex of the petrous bone. This might embrace the fourth nerve, which runs near this part, but could scarcely include the palpebral division of the third leaving every other portion intact unless the branch were given off unusually early. Of the two suppositions—a double lesion, or an abnormal distribution of the third nerve—the former is the more probable.

In January, 1871, a woman, aged 40, came under my care with paralysis of the left facial nerve, preceded by pain in the forehead, and accompanied by pain and tenderness of the mastoid process. This was removed under iodide of potassium in little more than a month. As to the nerves of the upper extremity, I have seen several cases, a few of which I may detail.

In November, 1864, a woman, aged 42, who lived an irregular life, though she did not acknowledge syphilis, came under my observation. She began to suffer a month previously from pain in the neck, down the right arm and forefinger, which had continued ever since. She was worse at night. The arm also was very weak and could not be raised. The neck was stiff and could not be turned in any direction without pain. There was

tenderness along the left side of the spine from the third to the seventh vertebra, and tenderness over the brachial plexus, probably from periostitis or inflammation of the fibrous structures involving the nerve roots. There was speedy relief and complete cure by iodide of potassium and one or two blisters. I saw her subsequently for a node on the femur.

. . . I may mention an example, an interesting one, of syphilitic paralysis of the lower limbs arising from affections of the nerves after leaving the spinal cord. It is a case of paralysis of the flexor muscles of the thigh and of the muscles generally of the leg and foot, loss of sensation over the front of the thigh and along the inner aspect of the leg. . . . The motor paralysis was in the sciatic area of distribution; the sensory mainly, though not exclusively, in the anterior crural area. The paralysed muscles were wasting, and the left limb was decidedly smaller than the right; and it was found later on that the psoas and iliacus muscles were weak. . . . The paralysis was clearly not spinal, since it first affected one leg only; and secondly, was both sensory and motor in the same limb, while any unilateral mischief in the cord is known to give rise to motor paralysis on the one side and sensory paralysis on the other. Throughout, its distribution was in the area of different nerves. Finally, muscular wasting indicated a severance of the nerves from the cord by the morbid process. I take the lesion to be one of syphilitic inflammation and deposit on the left side of the chorda equina. The man recovered completely under iodide of potassium.

LECTURE II

The Syphilitic Affections of the Spinal Cord

The range of symptoms producible by disease of the cord is not very wide. The tendency of most is to paraplegia; and to understand this we have only to remember that in some parts the cord is not thicker than the little finger, and that consequently a limited area of inflammation or a small tumour might involve a complete segment, which is all that is required to cut off the parts below from the volitional centres in the brain, and produce this form of paralysis.

Paraplegia is rather common as a result of syphilis (Bazin says that two-thirds of the cases are syphilitic), and if taken early and treated energetically it is usually curable. It is therefore very desirable that, if possible, distinguishing features of syphilitic paraplegia should be pointed out.

It has been given as one of the characters of syphilitic paralysis generally, and of syphilitic paraplegia, that sensation is usually not impaired, and that reflex action persists; but this, if it were true as a matter of fact, which I shall show is not the case, would not constitute a peculiarity. It is the rule in any form of disease of the nervous system, that the motor functions are more easily affected than the sensory. Even in nerves in which motor and sensory fibres are mixed up together, an injury, such as pressure, may destroy for some time the conductibility for motor impulses, while the only effect on the sensory functions is a temporary dysaesthesia. In experimenting on the cord this difference has been found to be even more marked: division of the white fibres constituting the motor tract being followed by distinct and definite motor paralysis; while in the case of the central grey matter, which is the channel for sensations, a slight bridge suffices to convey upwards the sensory impressions, and while there is any continuity of grey matter anaesthesia is not produced. It is again the rule in hemiplegia from cerebral lesion that motion is more frequently and persistently impaired than sensation.

It is not in individual symptoms, or in any arbitrary and unexplained difference in the phenomena, that we shall find the distinc-

tive characters of syphilitic affections of any part of the nervous system, but in a comprehensive view of all the facts of the case.

It will not occupy much time, and will conduce to clearness, if I enumerate the principal diseases which affect the cord, with their leading characters.

Myelitis—inflammation of the substance of the cord, if general, gives rise to a progressive loss of motor power from below upwards, with loss of sensation, preceded by tingling, the rectum and bladder and their sphincters sharing in the paralysis. There is little pain, at most a dull aching. Reflex action is abolished, and death occurs when the inflammation extends to the respiratory centres. Bed-sores come on early.

A local myelitis, involving a segment of the cord, causes paralysis of the parts below, motor and sensory (or motor only for a time), with loss of control over the evacuations. Reflex action will persist and may be exaggerated from concentration on the motor cells of the anterior nerve-roots of impressions which normally would have been partly transmitted upwards to the brain, and the paralysed limbs often start involuntarily. If there be pain it is usually due, not to the myelitis, but to some extraneous cause—as, for example, disease of the vertebrae.

Spinal meningitis is attended with pain both along the spine and in the limbs, provoked by movements, especially such as involve motion of the spinal column. There are painful startings, or tonic contractions of the limbs or muscles of the trunk, and sometimes severe tetanoid spasms; sensibility is usually intensified. Paraplegia comes on late, and gradually if at all. There is loss of power in the bladder; not usually in the rectum.

Spinal congestion, according to Dr. Radcliffe, gives rise to sudden incomplete paraplegia, varying in degree, without loss of sensation or of control over the evacuations, but attended with aching in the back and limbs; but there may be a rapidly-progressive fatal paraplegia which leaves only traces of congestion.

Other diseases are, sclerosis of the posterior columns and cornua, and of the anterior white columns and cornua, the former giving rise to locomotor ataxy, the latter to wasting palsy.

There may again be softening of the cord, which, if non-inflammatory, is not to be distinguished from local chronic myelitis; or it may be the seat of tumours.

Syphilis may give rise to myelitis or meningitis, or to the small gummata or local scleroses described by Dr. Moxon.

Myelitis.—The form of myelitis most frequently seen in syphilis is subacute or chronic inflammation of a segment of the cord; but, in my opinion, acute general or local myelitis may be caused

by syphilis, especially in the secondary stage. This is a point which cannot easily be proved. There is nothing peculiar in acute myelitis to indicate its cause; and, the disease being rare, it must be long before a sufficient number of cases can be collected in which it is associated with syphilis, even by observers alive to the possibility; while in the secondary stage a post-mortem diagnosis cannot be made so readily as in the tertiary period of the disease.

In 1859 I saw, in quick succession, two cases—one in the Hôpital du Midi under Ricord, the other under Trousseau in the Hôtel Dieu—of rapidly progressing paraplegia terminating fatally. In both there was general and advanced inflammation and softening of the spinal cord. Both the patients were young, and suffering from secondary syphilis in an early stage. Ricord, with characteristic caution, refused to pronounce the myelitis syphilitic, though it was not the first case of the kind he had seen in his wards. Trousseau was more disposed to consider syphilis as the cause of the inflammation.

Of acute local myelitis I give a case in which also the disease of the cord came on in the secondary period.

Paraplegia in secondary syphilis; acute myelitis of a segment of cord just above the lumbar enlargement; death from ulceration of bladder and extravasation of urine.

* * * * *

The *paraplegia* generally produced by syphilis is the result of disease slowly invading a segment of the spinal cord.

The course of symptoms is such as might result either from a limited chronic myelitis, or from softening, or from tumour.

The age at which syphilitic paraplegia occurs—namely, in early adult life or early middle age—excludes degenerative non-inflammatory softening.

Embolic softening, so far as I know, only occurs in connexion with a very general distribution of emboli, as in acute fatal chorea or as in ulcerative endocarditis, when the effects produced elsewhere predominate.

The absence of angular spinal curvature and of the symptoms which disease of the bones or cartilages of the vertebral column produces before the cord is affected, excludes the most frequent cause of local myelitis not depending on syphilis, and we are left to decide from the history or from collateral circumstances between syphilis and rheumatism or exposure to wet and cold or injury.

In syphilis the condition present is probably a small gumma around which inflammatory softening is set up, and this explains the slow, halting, and unequal progress of the paralysis.

Conversely, paraplegia, at first slight, remaining long at a given point or advancing very slowly, then suddenly worse, mending a little again, perhaps spontaneously, or at least without specific treatment, but again increasing, and so pursuing its course to absolute loss of sensation and motion, while reflex action persists, should excite a suspicion of syphilitic mischief and should lead to energetic treatment, whether corroborative evidence is forthcoming or not.

While it was still uncertain whether syphilis could give rise to disease of the nervous system, and the purpose of inquiry was to establish a fact in medical science, no caution could be too great in accepting evidence on the point; but the fact of causation once established we are justified in going in advance of positive knowledge in our treatment.

The following case is one of unusual interest, there being great impairment of sensation, and exaggerated reflex action, with loss of control but not of power. It might easily be taken for locomotor ataxy, but differs essentially from this affection in its symptoms, and, as I believe, in pathology.

Sensory paraplegia; loss of control but not of power; exaggerated reflex action.—The patient, a cabman, aged 34, married, but childless, was admitted into St. Mary's Hospital, March 7, 1873, for loss of power in the legs. He was a strong, stout, well-built man, with good colour, and large and firm muscles. He had, however, lost all his hair, not only from the scalp, but from the face, axilla, and pubes; the eyebrows and eyelashes also were gone. He had suffered from gonorrhoea ten years before, but had never had a sore on the penis; he had, however, had "measles" and a bad sore throat fourteen years previously, which lasted three months, and ample evidence of syphilis was afforded by large ulcerations of unmistakable character on both legs. The weakness of the lower extremities had been coming on for twelve months; he began to drag first the left foot, then the right, and the knees and feet gradually became numb. For three weeks he had been unable to walk.

On first seeing him I told him to rise, so that I might have the opportunity of noting the degree of paralysis and observing its character. He threw his legs off the bed without difficulty; but when the feet were put to the ground, and he endeavoured to stand, the attempt was completely defeated by violent spasmodic contraction of the legs, especially the right, which would have thrown him down had he not been caught and assisted back to bed. The movements continued for some time afterwards. He explained that he could stand and walk better with his stockings on than when the feet were bare, and still better in strong boots. The

difficulty obviously arose from excessive reflex action, which was more violent when the feet were in direct contact with the floor.

Lying in bed he could move every part of both lower extremities, and could resist forcibly any attempt to flex or extend any joint. The muscles were bulky and firm, and particularly well nourished. He had occasionally startings of the limbs at night. Sensation was greatly impaired in every part of the thighs and legs, whether tested by compasses or by pricking or pinching. The insensibility to heat was shown by the fact that he had large blisters on the soles of both feet, caused by a hot bottle which he had not felt. He had for three or four months had difficulty in micturition, had to wait some time before the flow began, and the urine came only in drops; it contained no albumen. Pulse and temperature normal.

To complete the account of the symptoms, it need only be added that, when he was able to stand or walk, he could do so equally well with the eyes shut or open, and had no particular difficulty in turning. He stood steadily with his eyes closed when he could only walk with the aid of two sticks. In walking, the legs were not shot out or jerked up vaguely as in locomotor ataxy, but carried along stiffly and wide apart, the feet scraping the floor in consequence of want of flexibility in all the joints, which were kept rigid by tonic contraction of the muscles generally. The hand placed on the thigh or calf when he walked found them hard and firm like those of an athlete.

Comparison with Locomotor Ataxy.—The phenomena of this case are worthy of study and discussion. They were clearly not those of the condition known as locomotor ataxy, although they would be covered by the term. The mode of progression, the power of standing with closed eyes, the absence of pains in the limbs, exclude at once sclerosis of the posterior columns of the cord, which is the morbid change giving rise to locomotor ataxy, and there are other differences of equal significance.

I cannot fully convey to you the precise grounds on which I come to the conclusion at which I have arrived without reference to the functional mechanism of the spinal cord, as I understand it. The anterior grey columns consist demonstrably of the nuclei of the anterior or motor roots of the nerves fused together; the posterior grey columns in the same way of the nuclei of the posterior nerve roots (this is inferred rather than distinctly proved). The antero-lateral white columns are the downward channel for motor impulses from the corpus striatum to the motor nerve-nuclei; the central grey matter is the channel for sensory impressions upwards from the posterior nerve-nuclei to the thalamus; decussation of the

fibres or cells constituting the sensory channel taking place one by one in the cord.

Now, Brown-Séquard has shown that injury to the central grey matter will intercept sensory impressions without interfering with motor power, and this is exactly what had occurred in the case under consideration, and I concluded that a syphilitic tumour existed in the very axis of the cord.

But the posterior and anterior nerve-nuclei together constitute the apparatus for reflex action, and there is in the cord an independent power of co-ordination—that is, of orderly combination of muscular movements for a given purpose, the mechanism for which consists in the linking together of the nerve-nuclei of different parts of the cord. It would have been difficult to say, *à priori*, whether such co-ordinating fibres would connect together anterior or posterior nerve-nuclei; but it has been found experimentally that it is through the posterior nuclei that co-ordination is effected, the fibres which effect it running in the posterior white columns. I am of opinion, however, that there is an association of the anterior nerve-nuclei of different segments, as I find no other explanation of the peculiar arrangement of the fibres of the anterior white columns, and I think it serves some such purpose as the combination of the action of one fore-leg with the hind-leg of the opposite side in dogs, such association explaining the fact that injury to the corpus striatum does not occasion hemiplegia in dogs.

Let me, however, compare this case in which I suppose a segment of the central grey matter to be damaged, with locomotor ataxy, in which the posterior white columns are affected. In both we have impairment of sensation, with comparatively little loss of motor power, but with loss of control. In locomotor ataxy there is destruction of the fibres in the posterior white columns, which convey the impressions from one part of the cord to another, and bring about the delicate adjusting movements which insensibly maintain the balance of the body, and the patient is unable to stand with the eyes closed. My patient stood perfectly well. The loss of balancing power, then, in locomotor ataxy, is not simply due to impaired sensation in the feet, as has been maintained by Dr. Allbutt.

Again, in locomotor ataxy, motor impulses from the cerebrum for the act of walking arrive at certain nuclei in the lumbar swelling of the cord; in consequence of the disease in the posterior columns, these impulses are not diffused along them to the different segments of the cord which co-operate in the production of the harmonious movements of the limbs, but are con-

centrated upon the nuclei on which they impinge, throwing them into excessive and disorderly action and causing the jerky gait. This was not the case with my patient; but, on the contrary, the impressions received by the feet in contact with the floor, instead of being partly transmitted to the brain for the communication of sensation, were, in consequence of the interruption in the upward channel, concentrated upon the cord, but distributed to the different segments by the posterior columns, producing the excessive but co-ordinated reflex contraction of the muscles of the limbs, which restrained or restricted their obedience to the will.

A small tumour such as was indicated by the symptoms to be present in the axis of the cord was just the kind of change to which syphilis might give rise, and which scarcely any other disease would produce. This is one of the examples, then, in which the symptoms alone would almost warrant a diagnosis of syphilis, without any corroborative evidence.

The progress of the case was satisfactory. I began the administration of iodide of potassium in doses of ten grains, with carbonate of ammonia and sarsaparilla, carrying it eventually to twenty-five grains three times a day. In a few days the nocturnal startings were less troublesome, then sensation began to return, and he passed water better. He walked with the aid of two sticks on April 3, and on the 26th with only one, passing urine now quite readily; sensation also was much better, but not normal; the ulcers on the legs had healed. About this time faradisation was ordered. He left the hospital on May 2, after a stay of less than two months. On the 27th he drove a hansom to the Derby. He continued to take the iodide (fifteen grains). . . .

Meningitis.—Spinal meningitis is far less common than myelitis, and I have met with no acute case distinctly traceable to syphilis. A patient, however, was under my care in September last, suffering from severe pain in the back and limbs, made worse by movements, with feverishness, for which the cause assigned was a fall in which the back was injured. The fall, however, had happened a month before, and he had since worked at his trade without pain or inconvenience. An explanation of the pain soon appeared in an outbreak of syphilitic eruption, and it was removed by a mercurial course. I have now (January 15) in hospital a patient recovering from a subacute attack, who had previously suffered from paraplegia, and whose case I have already given. She left the hospital well on August 9, and remained free from symptoms till when she began to suffer from "rheumatism," as she supposed. There was, however, no swelling of the joints or fever, and the pain was pro-

voked by movements of the limbs or spine, and had the character of pain caused by spinal meningitis. She began also to lose power again in the lower extremities. The pains were at once relieved by iodide of potassium.

One of the worst cases of syphilitic affection of the nervous system which I have ever seen, and which ultimately resulted in insanity, began with spinal meningitis. The patient was under the care of Mr. Walter Coulson, with whom I saw him, and the case is partly related in his treatise on syphilis. When I first saw him, in August, 1866, he was complaining of rheumatism — obviously spinal meningitis — which completely crippled him. Every movement gave him pain, but especially the first efforts after being for a time in one position, so that it was distressing to watch him rise from a chair. He carried himself stiffly, was unable to stoop or to turn the head; in order to look round he had to turn the whole body. There had previously been tertiary manifestations, and the meningitis yielded to iodide of potassium, leaving the patient, however, with a stoop. To this was probably attributable also sexual impotence, from which he was found to suffer afterwards.

Syphilitic Affections of the Medulla Oblongata and Pons.

The intra-cranial prolongation of the spinal cord forming the medulla oblongata and pons Varolii is complicated in its structure and function, not only by the separation of the nerve-nuclei from each other in accordance with the specialization of their functions, but by the change in the relations of the grey matter and tracts of white fibres with each other, the decussation of the motor tracts, and the connexion of the cerebellum with the spinal axis which is here effected. The consequences of lesions here, therefore, are more varied, and the causes which may give rise to them are increased in number; these parts, for example, may be affected by disease in the cerebellum or by aneurism of the basilar artery.

The increased dimensions of the spinal axis, moreover, permit of unilateral limitation of a morbid change; and this is not uncommon, although the two halves are fused together; while unilateral mischief is rarely seen in the cord, which is almost completely divided longitudinally by the fissures. Possibly, however, the vascular membrane which dips into the fissures is more efficient in communicating a morbid change than nervous structure.

The general results of disease in the medulla and pons are, some interference with the sensory or motor tract proceeding from the cord to the sensori-motor ganglia, together with disturbance or abolition of the function of one or more of the nerve-nuclei. It is seldom

that we can come to the conclusion that there is disease here from sensory or motor paralysis of the limbs alone, or from affections of the nerves which have their origin in the intra-cranial part of the spinal axis alone, although this may be done occasionally when two nerves are simultaneously paralysed, the nuclei of which are conjoined or in close proximity, while the nerves take a different course after their emergence from the centre, as in the case of the sixth or seventh.

The grouping of symptoms arising from the inclusion in a diseased area of several nerve-nuclei may be very varied. Usually the symptoms afford the means of making a very precise localization of the lesion by the application of anatomical and physiological knowledge. Of course disease here is attended with great danger to life, the slightest interference with the reflex centres of the respiratory or cardiac movements being fatal. Sudden death, therefore, is common.

The nature of the morbid change will be arrived at by considerations such as those made use of in determining the probable cause of paraplegia. A slow but irregular progress of the affection will favour the hypothesis of syphilis, but the morbid changes resulting from syphilis do not specially affect the surface of the medulla and pons, according to Dr. Moxon's dictum, but rather the substance. The progressive labio-glossolaryngeal paralysis, which is the counterpart of locomotor ataxy, is not syphilitic; but it is quite possible for syphilitic change to involve the same parts of the medulla and give rise to analogous or even identical symptoms. The course of the disease will, however, be different.

Case I.—*Paralysis (hemiplegic) of left face; lateral deviation of eyes to right; impairment of articulation and deglutition; loss of sensation in right face; sudden death; syphilitic tumours in pons and medulla. . . .*

Case 2.—*Slight right hemiplegia of gradual access; impaired articulation; peculiar affection of respiration; sudden death.*—Ross C., a sweep, aged 39, became an out-patient at St. Mary's Hospital on January 30, 1865. He had gradually been losing power in the right limbs for a year, and the leg, hand, face, and tongue trembled. His speech was slow and his utterance thick. The right pupil was smaller than the left. He was "very heavy for sleep," and would often fall asleep in his chair, and while sleeping his wife said he turned blue, and at length woke up suddenly, appearing bewildered, and often laughing. The articulatory defect was greater than was warranted by the degree of paralysis of the limbs, and was of a kind which suggested lesion of the medulla as its cause. The partial asphyxia which occurred during sleep also pointed to the

medulla as the seat of some disease which made the reflex respiratory movements insufficient, and rendered supplemental voluntary respiration necessary.

He had had syphilis, and iodide of potassium in eight-grain doses was given three times a day, and a Plummer's pill at night. He improved considerably, his speech became less thick, he regained almost full power in his right limbs, and was able to resume work. On March 9 he paid his last visit to the hospital, and was remarked by his wife to be in very good spirits on account of his improvement. In the course of the day he took beer to excess with a friend in celebration of his recovery, went home, lay down on his bed, fell asleep and was found dead. I heard nothing of this till three or four weeks later. A post-mortem examination had been made, and an inquest held, the cause of death assigned being heart disease. I afterwards saw the medical man who had made the examination, and found that the opinion given was an inference from the mode of death, and was not based on definite change in the heart. No morbid appearance had been found in the brain, but there had been no special examination of the pons and medulla.

Syphilitic Affections of the Cerebellum.

The symptomatology of disease of the cerebellum is singularly obscure, corresponding, however, in this respect with the state of our knowledge of the physiology of this nervous centre.

I still adhere to the theory of its action, which makes it the seat of the higher and more complex co-ordinations of movements. It is not the sole organ of muscular co-ordination, as was at first inferred from the experiments of Flourens. The spinal cord co-ordinates in a distinctly purposive manner movements which respond to tactile or other cutaneous impressions, but when movements have to be guided by vision, some special and more complex apparatus is needed to bring muscular action into relation with visual impressions, which are so far removed in character from tactile impressions (the immediate guides of muscular actions), and are correlated with them only by inference and experience.

This is essentially, though in a superficial and imperfect way, the view held by Mr. Herbert Spencer, and repeatedly expounded and illustrated by Dr. Hughlings Jackson, namely, that the cerebellum is to space relations what the cerebrum is to time relations. Dr. Ferrier's interesting experiments, which appear to make of the cerebellum simply a motor centre for the movements of the eyes, receive no support from the facts of

pathology, and his results will probably find some interpretation more consistent with these.

As in the cerebral hemispheres, so in the cerebellum, there may be extensive disease without obvious symptoms, or the symptoms may be such as will indicate only the existence of intra-cranial mischief, but afford no indication whatever of the seat of the lesion, viz., pain in the head, vomiting, and blindness; pain, however, being more constant as an accompaniment of cerebellar than of cerebral disease.

The special symptoms of disease of the cerebellum, when such exist, are a peculiar staggering gait and a vague, purposeless character of the movements generally, together with a marked loss of vigour and energy. A want of control and co-ordination of muscular actions is evident, but it differs from that due to loss of spinal co-ordination, and it is not increased by closing the eyes. In the later stages, evidence of pressure upon the medulla, or of general intra-cranial pressure, may be superadded, due to effusion into the cerebral ventricles. In the cases of disease of the cerebellum which I have watched to a fatal termination, and in which I have made post-mortem examinations, the cause has not been syphilitic, and in the case I am about to relate, which I considered to be one of syphilitic affection of the cerebellum, no examination was made.

Headache; fits of uncertain character; hemiopia; later slight right hemiplegia; sudden death. . . .

LECTURE III

On Syphilitic Affections of the Brain, Meninges, and Cerebral Arteries

I have still to bring before you the affections which result from the effects of syphilitic disease upon the brain, its membranes, and blood-vessels. So numerous are these and so important that I could well occupy in even a brief account of them more than the number of hours allotted to the entire series of lectures; and they are so varied and protean that I might be excused if I took refuge in a merely symptomatic classification of them. But even were I to bring them before you under the heads of paralytic and convulsive affections, and affections of sensation or of the intelligence, I should find myself in a difficulty of another kind. In many of the cases there are both convulsions, paralysis and mental derangements; and these could only be assigned to one or other group in an arbitrary manner. Moreover, any of these symptoms may arise in one case from one cause, in another from some lesion entirely different both in seat and character, and one classification would fail altogether to aid either in prognosis or treatment.

A real diagnosis implies the identification of the part which has suffered injury and a recognition of the morbid process which has inflicted it. We can form a rational estimate of the chances of recovery and direct intelligent efforts for its attainment only when a distinct and definite idea has been formed respecting the disease we have to treat, and it is only when this has been done that we can profit by our mistakes. Let us have error rather than confusion.

I must here again briefly enumerate the morbid changes which syphilis induces in the brain. You will remember that the characteristic is a tendency to the exudation of a particular kind of plastic material which may be diffused in the membranes at the base of the brain or over the hemispheres, or may take the form of a distinct tumour, which will frequently have a great resemblance externally to malignant growths. The "habit of locality" of the tumours, as well as of the diffused exudation, is to affect the surface, although gummata or syphilomata may be found in the substance

of the brain; usually, however, in the more vascular parts—the grey matter of the corpora striata or thalami.

In the diffuse form we may have the membranes adherent to each other and to the convolutions by means of the firm plastic material described in my first lecture; and, as a result, the vessels of the pia mater are occluded, the supply of blood to the peripheral grey matter is diminished, and this undergoes atrophic change of some kind; or small indurations may invade the nervous structures from the membranes. Disease of the surface grey matter of the hemisphere may give rise to convulsions or paralysis, or the most varied intellectual or moral disturbances, according to the particular set of convolutions affected, and the nature and rate of progress and stage of the morbid process. This it is—the tendency to affect the membranes, and the varying intensity of the inflammation—which makes syphilitic affections of the brain so multiform.

The order in which I propose to consider the different cerebral affections resulting from syphilis is as follows: Syphilitic Epilepsy, with cases; the graver results of Syphilitic Disease of the Membranes, first at the base, then over the hemispheres; Syphilomata or tumours; Cerebral Disease in Infantile Syphilis; Syphilitic Thrombosis of Cerebral Arteries.

Syphilitic Epilepsy.

As disease of the surface of the hemispheres commonly gives rise to convulsions, it is not surprising that convulsive seizures are among the most common symptoms in syphilitic disease of the brain. Attacks of convulsions may usher in a train of grave disorders of nervous functions, or may be one among many concurrent phenomena, or may form the only important symptoms. They may be produced by tumours growing either from the bones or dura mater or in the pia mater, or in the substance of the hemispheres, when these reach the surface; or by diffused exudation in the pia mater, or by thrombosis at the time of its occurrence and during the consecutive changes; or by slighter changes affecting the nutritive vigour of the hemispherical grey matter.

They are generally late manifestations belonging to the tertiary period of syphilis, though I have seen syphilitic epilepsy within a few months of the first evidence of constitutional affection. It is probably one of the slighter meningeal affections which is present in those cases of so-called syphilitic epilepsy in which the convulsive or epileptiform seizures are the prominent symptoms throughout, and not simply the precursors of graver forms of disease. It is probable, again, that syphilitic disease of the arteries not giving rise to extensive thrombosis may produce sufficient interference

with the cerebral circulation to impair the nutritive vigour to a degree which will permit of the irregular discharge of nerve force, just as we see sometimes in the epilepsy of advanced life.

In epilepsy proper, or idiopathic epilepsy, there is frequently absence of any lesion to which it can be attributed, and it might be that in syphilitic epilepsy no appreciable lesion would in some instances be found. Authorities, again, are still at variance as to the nervous centre in which the paroxysms start. I have not had the opportunity of examining post-mortem a case in which the epileptiform convulsions had been the only nervous symptom due to syphilis, or met with a description of such a case; but although experiments seem to show that disorder of the cortex cannot be the only cause of convulsions of the kind seen in epilepsy, it is certain that it is a frequent and important cause; and the associated symptoms make it certain that in syphilitic epilepsy the cerebral hemispheres are affected. These associated symptoms, together with the age at which the periodical convulsions come on and a syphilitic history, constitute the peculiarities which serve to distinguish syphilitic epilepsy. Before entering upon this point I will relate a few cases. The first to be given was under observation for several years.

Epileptiform convulsions; frequent attacks of petit mal, mental enfeeblement, at one time mania, together with syphilitic keloid, and tumours in the tongue. Recovery under large doses of iodide. . . .

CASE 2.—*Frontal node; epilepsy; attacks of giddiness and trembling, with mental depression. Recovery under iodide. . . .*

CASE 3.—*Syphilitic sore throat; violent epileptiform seizures; pain in the head; hesitating speech; weakness in the legs. Recovery. . . .*

CASE 4.—*Nodes and other tertiary manifestations; epilepsy; headache; giddiness strange feelings. Recovery. . . .*

Looking over my notes of old cases of epilepsy, I find several which, in the light of subsequent experience, I am now inclined to set down as of syphilitic origin, notwithstanding the absence of ascertainable syphilitic antecedents.

I may now sum up the features of epilepsy symptomatic of syphilitic disease of the brain by which it may be distinguished from ordinary epilepsy.

As to the convulsive attacks themselves, there is nothing distinctive either in their character or frequency, or in the time of their access—i.e. whether nocturnal or diurnal. It is difficult to obtain a reliable description of a convulsive seizure, and the opportunities of watching an attack are very rare; but after careful questioning of witnesses in cases of idiopathic and syphilitic epilepsy, I have

come to the conclusion given, which is, moreover, that generally accepted. If this statement can be qualified in any way, it would be the greater irregularity of the intervals between the fits.

An important distinction, however, exists in the fact that the intervals between the convulsive paroxysms due to syphilitic disease of the brain are not intervals of perfect health and freedom from nervous disorders. On the contrary, if there are not nocturnal headaches or osteocopic pains or sleeplessness, such as are caused by syphilis independently of disease of the brain, and diagnostic of syphilis, there may be frequent attacks of petit mal, often many times a day, or of convulsive twitchings of the eyes or of a limb, or merely of vertigo or of faintness ; or there may be a state of extreme and unaccountable nervousness and apprehension.

Again, we are often put on the track of syphilis by the age at which the epileptiform attacks first come on. It is matter of universal experience that true epilepsy is a disease of early life. If the predisposition, hereditary or otherwise, exists, the disease develops itself before the changes attending the full evolution of the sexual organs are completed. Dr. Russell Reynolds gives the following statement of the age at which epilepsy began in 172 cases : Under ten years of age, 19 ; between ten and twenty, 106 ; between twenty and forty-five, 45 ; over forty-five, 2. But of the forty-five cases in which the disease began between twenty and forty-five years of age by far the larger part began about the age of forty ; so that in early adult life there is almost complete immunity from epilepsy. Now it is just at this period that the epileptiform seizures due to syphilis most frequently come on. When, therefore, a young adult begins to have convulsions of epileptiform character we may at once suspect syphilis, and the suspicion will be strengthened if there are other nervous phenomena, and may be converted into certainty by evidences of past syphilis—nodes, perforations or cicatrices in the throat, white marks on the tongue, and pigmented scars at different parts.

Once let it be ascertained that epileptiform attacks, not accompanied by other evidences of cerebral disease than such as I have enumerated in the cases I have given, are due to syphilis, and the prognosis is most favourable. There is always a liability to relapse, but I have known almost complete immunity from all symptoms to be enjoyed for more than ten years.

Syphilitic Disease of the Meninges.

The cases in which the membranes are more gravely affected, and the surface grey matter invaded or involved, or deprived of blood, present an inexhaustible variety of symptoms from different

combinations and successions of convulsive and paralytic affections and intellectual derangements. Speaking generally, paralysis of cranial nerves and of the limbs, gradual in their mode of access, are characteristic of disease about the base of the brain; convulsions and mental affections, of disease on the convex surface of the hemisphere.

No strict line of demarcation can be drawn between the cases in which there is extensive exudation in the membranes, and those in which the morbid process results in the formation of distinct tumours. In the former, the deposit frequently here and there takes the form of a nodule, which projects into the brain substance, and a syphiloma is accompanied by or sets up changes in the adjacent part of the meninges. I shall not, therefore, attempt to lay down distinctions between diffused and localized syphilitic exudations; but, in the brief comments I may make on the cases, show how I have come to my conclusions as to the character of the lesion. The cases I give first will be illustrations of disease in the membranes of the base of the brain.

CASE 1.—*Complete paralysis of the right third and sixth, and probably fourth nerves, and partial motor and sensory paralysis of the entire opposite half of the body.* . . . Here there could be no doubt as to the seat of the lesion. It could only be at the base of the brain, round the right crus cerebri, involving the nerves of this side in the intra-cranial part of their course, and affecting the crus which here contains the fibres, sensory and motor, of the entire opposite half of the body. The character of the morbid change was, with almost equal certainty, syphilitic inflammation attended with gummatous deposit in the pia mater.

He was discharged apparently well, but with paralysis of the right sixth nerve persisting. . . .

CASE 5.—In November, 1872, I saw with Dr. Langmore a patient who was the wife of a man who at the time had severe syphilitic rupia. She had had one miscarriage, and no living children; no syphilitic manifestations. Six months before I saw her she had had paralysis of the right side of the face, and numbness in the right arm, which recovered in six weeks. She had been complaining of numbness round the mouth and in the left arm, and she suffered from intense frontal headaches, which generally came on at 3 a.m. Sensation was found to be impaired in the left arm, and the grasp was weak. She walked well. Sensation was also deficient over the lower part of the right side of the face in the area of distribution of the two lower divisions of the fifth, the anaesthesia transgressing the median line about the mouth. The forehead, the tip of the nose, and the tongue were not affected. The sight was not good,

and she was unable to read long; the optic discs were pink, striated by vessels, and the retinal veins were large, but there was no appearance of swelling, and the discs were well defined. The patient was taking iodide of potassium in doses of 6 grains, and it was agreed that the dose was to be gradually increased to 24 grains. This failing, mercury was to be tried.

Here the cross paralysis, incomplete anaesthesia in the two lower divisions of the right fifth nerve, and weakness and impaired sensation in the left arm, pointed to a lesion near the apex of the petrous bone, or the adjacent part of the sphenoid, involving the two branches of the trigeminus after their departure from the Gasserian ganglion, and the crus cerebri. Had the entire fifth nerve been affected, the seat of the disease might have been the pons. The severe pain led me to conclude that the disease had its starting point in the bone or dura mater, and I anticipated difficulty in affording relief. I learn, however, that she improved greatly under the iodide of potassium. She is liable to headaches, but there is now no paralysis.

Syphilomata or Tumours.

Among the most interesting examples of syphilitic disease of the brain are those in which the morbid deposit takes the form of a distinct tumour. The symptoms common to nearly all cerebral tumours, when any symptoms at all are present, are severe pain, vomiting and double optic neuritis; the pain being fixed in seat or radiating from one point, but variable in intensity at different times. Cases, however, are on record in which tumours have attained a large size without giving rise to abnormal phenomena of any kind; and although the occurrence for any long time of the three symptoms mentioned would be almost conclusive of tumour, the absence of one or other would not necessarily be conclusive to the contrary. Superadded to these general symptoms may be hemiplegia, motor or sensory, if the central ganglia are involved; or paralysis of individual nerves, if the tumour is situate at the base of the brain; or convulsions, if the surface grey matter of the hemispheres is affected.

Optic Neuritis.—The symptom of greatest importance is unquestionably the double optic neuritis; and, as Dr. Hughlings Jackson in particular has pointed out, it may for some time be the only symptom, or, if not quite alone, may be associated with symptoms so slight as to have no significance independently of it.

We must not wait till vision begins to suffer before examining the eyes. It is with constantly renewed astonishment, notwithstanding the frequent illustrations brought forward by Dr Jackson, and

exhibited to this society and elsewhere, and my own experience, that I see the extensive changes in the disc compatible with good vision.

We are as yet in uncertainty as to the immediate mode of production of optic changes, and of the chain of causation by which they are connected with tumours. It may perhaps be taken as certain that one way in which changes are produced is by increased intra-cranial pressure; and it appears probable, moreover, that this intra-cranial pressure operates through fluid being forced along the sheath of the optic nerve, between its two layers, which bulges out the sheath near the eye and compresses the nerve and its vessels. But the optic changes may be entirely absent or, having appeared, may subside, when we have every reason to suppose, from examination after death, that intra-cranial pressure must have existed, and may be present when we can see no cause of intra-cranial pressure. If the existence of fluid in a position to be carried into the optic sheath is a condition required for the production of strangulation of the nerve, the absence of effusion in the membranes at the base of the brain may explain some cases in which optic changes have not appeared.

But it is not even a settled point whether there is or is not a distinction between ischaemia of the disc, a vascular strangulation or obstruction due to intra-cranial pressure, and neuritis descendens, an inflammation propagated to the disc by the continuity of the neurilemma with the pia mater; or, admitting the distinction, whether the difference can be made out by ophthalmoscopic characters. Till this is decided we cannot so much as pass the threshold of the inquiry as to the mode of production of optic changes in diseases of the brain. If I were not held back by the doubts of men such as Dr. Hughlings Jackson and Mr. Hutchinson, whose opinion in this matter I hold to be far higher than my own, I should consider my own experience warranted me in following Dr. Clifford Allbutt in recognizing a difference between optic ischaemia and neuritis.

While we cannot explain satisfactorily the mode of production of optic changes, their clinical associations and significance are better known. They commonly, almost constantly, accompany tumours in whatever part of the brain they may form, and perhaps more constantly syphilomata than any others; they usually accompany abscess; very frequently meningitis, especially inflammation of the membranes at the base of the brain; rarely haemorrhage, unless as a consequence of consecutive inflammation; rarely embolism or thrombosis, though in one case of thrombosis I have seen the most marked optic ischaemia. They do not accompany the mole-

cular changes—inappreciable to the naked eye, and, as a rule, even by the microscope—which give rise to epilepsy or chorea.

Syphilomata usually give rise both to general symptoms and to such as aid in fixing the locality, and I do not remember to have read of a case in which an unsuspected syphilitic tumour has been found after death. This is no doubt a consequence of the fact that syphilomata affect either the surface of the hemispheres, or, if they form in the substance, it is at vascular parts such as the central ganglia.

Jacksonian Epilepsy.—I must again trust to my cases to illustrate the effect producible by syphilitic tumours; but I must notice more particularly a form of affection to which Dr. Hughlings Jackson has specially called attention as frequent in connexion with syphilis, and in some sort characteristic of syphilitic cerebral disease. The prominent feature in these cases is unilateral convulsion, unattended for the most part with loss of consciousness. The convulsive movements may vary in degree from a mere twitch or slight stiffening to the most violent agitation, and may be accompanied or preceded by sensations of various kinds.

Usually the starting point is constant in a given case, and very frequently this will be the thumb and index finger. Beginning here, the agitation may in one attack be confined to the upper extremity, and there may be no loss of consciousness; at another time it will invade the entire lateral half of the body, travelling up the arm to the shoulder and face, and down the leg, becoming bilateral where the nerve nuclei of the two sides are associated; or sometimes the arrival of the agitation and accompanying sensation at the head or face may be the signal for general convulsions and loss of consciousness. If the convulsive movements begin in the foot or face a similar course may be followed, and after the unilateral convulsions the limbs which have been affected may be left paralysed for a time from exhaustion of the nerve force. When the hemispasm, as it has been called, is on the right side, and especially when the starting point is the face or tongue, temporary loss of speech is very common.

Sooner or later, and very often early, optic neuritis comes on. Dr. Jackson has shown that this hemispasm is due to disease in the convolutions of the opposite hemisphere, almost always near the fissure of Sylvius, and has shown that particular convolutions are involved according as the convulsions begin in the hand, foot, or face; thus extending our knowledge of the localization of function in the cortex of the brain, and giving occasion for the experiments of Dr. Ferrier, which have deservedly excited universal attention. Dr. Jackson has usually found the disease in these cases to be a

syphilitic tumour, but the symptoms are of course determined by the situation and not by the nature of the growth. The frequency, however, of syphiloma in these cases will, in cases of doubtful character, be a reason for entertaining the hypothesis of syphilis as a provisional diagnosis.

I do not find many examples of unilateral convulsions in the large collection of cases of syphilitic cerebral mischief made by Zambaco, Gros, and Lancereaux, one reason being that observers have not been duly aware of the importance attaching to them. The latter authors giving the situation of syphilitic tumours in the brain, find in one case the whole cerebral mass infiltrated; in one, disseminated exudations in different parts; in six, tumours in anterior lobes; in three, in the middle lobes; in three, in the corpora striata; in three, in a great part of the hemisphere. Some of these must certainly have been Dr. Jackson's cases.

Cerebral Disease in Infantile Syphilis.

Cerebral affections have not been frequently met with in infantile syphilis, but a few cases are on record, and it is extremely probable that many have been considered to be examples of tubercular meningitis. If no post-mortem examination had been made in the case I first relate, or if the post-mortem evidence had not been so conclusive, I should have been unable, either from the symptoms or from the history, to pronounce it syphilitic. Additional interest is given to this case by another with which I am opportunely favoured by Dr. Heywood Smith, in which the mode of access of the meningitis was strikingly similar. Dr. Smith's patient has happily survived the attack.

Syphilitic disease of brain and liver in a child; convulsions, left hemiplegia, coma; induration of right cerebral hemisphere and part of thalamus; acute meningitis.—A child, 3 years of age, was brought to me at St. Mary's Hospital in May, 1873, suffering from acute cerebral symptoms. The mother had never been well since her marriage; had only had one other child, and this died at the age of five weeks from atrophy; no miscarriages. This little patient was small, had always been delicate, had cut her teeth late, and had already lost some of them. The fontanelles were still open, but there was no other evidence of rickets; had only walked three months, but talked early; was extremely sharp and precocious, and liable to fits of excitement, in which nothing would pacify her. The child was said to have had a series of falls three weeks before, and to have knocked her head, but had apparently been none the worse till two days before her admission, when she became unconscious, screamed and kicked, and the convulsion of which this

description was given lasted an hour and a half, after which she recovered. She remained conscious, however, only a few hours, and it was observed the left limbs were paralysed.

She was unconscious when admitted, moaned much, and at times screamed. The face was pale; the eyes half open, and always turned to the left; the pupils small; no facial distortion. Left arm and left leg partially paralysed, the leg apparently more than the arm, slightly rigid; the thumb drawn into the palm. The right limbs, and especially the leg, were continually in motion. Pulse frequent and weak; swallowed food well; bowels not open; urine passed in bed. I failed to obtain a satisfactory view of the optic discs. She remained in much the same state for two days, when another attack of convulsions came on, affecting chiefly the right limbs, which were afterwards more quiet, but there were frequent convulsive attacks in which the limbs jerked less violently and the hands and feet became bent and rigid, so remaining even after death. During the last few hours of life the temperature rose rapidly to 105°.

On post-mortem examination the lungs were greatly congested, but no tubercle was found in them or in any part of the body, and the abdominal and thoracic viscera were normal, with the exception of the liver, which was small, pale, and presented on its surface several depressed cicatrices of different sizes, with vascular ramifications. They were not deep and the capsule was not greatly thickened; the lobules were wasted at these parts.

Brain: When the dura mater, which presented nothing abnormal, was removed, intense meningeal injection was seen at the posterior part of the hemispheres, especially on the upper surface, but also on the inner and lower aspect; it extended into the sulci. There was undue softness of the posterior part of the right hemisphere about the annectent gyri; almost black redness of the apex of the left occipital lobe, which was of leathery hardness. Nothing abnormal at the base or in the Sylvian fissures; no tubercle anywhere. On section of the brain there was seen great hyperaemia of its substance, especially posteriorly; the red points and striae were excessively numerous, and there was a general dusky pink staining. A patch of red and softened brain-substance was found near the surface of the right hemisphere about the annectent gyri. The ventricles had contained an excessive amount of fluid; the surface of the corpora striata and the ventricular walls generally presented unusual vascularity, especially posteriorly; in the right corpus striatum were one or two vascular patches, but its consistence was normal.

The principal change was in the left occipital lobe, which was hard

and shrunken. The induration was bounded by the occipito-parietal and calcarine fissures, and involved therefore the cuneiform lobule. The pia mater was of a deep-red colour, almost black, and adherent. The grey substance had undergone atrophy; the white was firm and tough like leather; the posterior cornu of the lateral ventricle was enclosed by this indurated white substance, but from the interior presented no abnormal appearance. There was superficial induration of the posterior part of the left optic thalamus, and some induration of the superficial transverse fibres of the pons. Cerebellum normal.

CASE 2.—*Symptoms of tubercular meningitis with an hereditary syphilitic origin.* Recovery under mercury and potassium iodide. . . .

Syphilitic Thrombosis of Cerebral Arteries.

Varied and important as are the effects of syphilitic disease in the membranes and substance of the brain, not less so are the results of syphilitic disease of the cerebral arteries. Here, again, as in every part of the subject, I have to acknowledge my indebtedness to Dr. Hughlings Jackson, who has led the way in this, as in many other investigations, and has repeatedly insisted on the importance of recognizing the effects of syphilitic disease in the arteries.

The arteries of the brain in syphilis are frequently attacked by inflammation, usually beginning in the outer coat. This may lead to thrombosis, which cuts off the supply of blood, and produces the results now known to follow this event. The effects are, first an accumulation and stagnation of blood in the capillaries in the area of distribution of the vessel blocked, and unless collateral circulation can be established there will be subsequent softening. The symptoms will depend upon the part fed by the artery which is obstructed. I am disposed to think they are more varied than those produced by embolism, since a fragment carried from the valves of the heart or from the aorta appears to find its way into certain vessels (notably the left Sylvian artery) in preference to others, while syphilitic thrombosis may occur anywhere. Syphilitic thrombosis must be of very frequent occurrence, judging from my own experience and from the number of cases collected by Zambaco and Lanceraux, but set down as examples of inflammatory softening.

CASE 1.—*Left hemiplegia affecting chiefly face and upper extremity; onset without loss of consciousness; convulsive attacks followed by mania; peculiar loss of intelligent use of hands without loss of power; syphilitic disease of arteries; thrombosis of right middle cerebral; softening of convolutions near end of fissure of Sylvius, etc.* . . .

CASE 2.—*Right hemiplegia with tonic contraction of paralysed*

limbs, relaxing during sleep ; access sudden, without unconsciousness, and attended with temporary aphasia. . . .

(Several other cases are also related.)

A few words remain to be said on *the diagnosis, prognosis, and treatment.*

The considerations involved in the *diagnosis of syphilitic disease of the nervous system* are too numerous and elaborate to be resumed in the time which remains at my disposal. We have, on the one hand, to guard against the conclusion that whatever happens in a person who has suffered from syphilis is necessarily due to the disease ; and, on the other, to avoid being misled by the absence of an acknowledged syphilitic history or of traceable syphilitic antecedents.

The period of life at which the nervous affection comes on is a great guide. In old persons, except in very obvious cases, we should arrive at a diagnosis of syphilis only after exclusion of other and more common causes of disease of the nerve-centres. In young adults syphilis would suggest itself early, unless there were heart disease or disease of the kidneys. Our chief aid in diagnosis, in addition to evidence of syphilitic disease in other parts, which must be carefully looked for, will be the antecedent or associated symptoms which we have learnt by experience to connect with syphilis—headache, with nocturnal exacerbations, sleeplessness and irritability. The gradual and irregular mode of access, except in the case of thrombosis, is, again, suggestive of syphilitic disease ; and convulsions are very common.

In *the prognosis* we have always to bear in mind the liability to relapse. Occasionally we see recoveries which are apparently complete and permanent ; frequently, I think, when the symptoms have been only epileptiform attacks, and the associated nervous disturbances enumerated in speaking of syphilitic epilepsy ; sometimes when there has been evidence of greater mischief ; but in a large proportion of the cases the patients will enjoy immunity from similar or more serious symptoms only on condition of perseverance in the employment of the remedies.

The chief considerations which bear on the prognosis are the duration, nature and seat of the lesion. As to duration, the longer the mischief has existed, the more likely are its effects to be permanent ; for although syphilitic exudations and growths are singularly amenable to the influence of remedies, if they are allowed to remain for any length of time they destroy the structures in which they are lodged ; this is more particularly important in the spinal cord, in which a very limited lesion will involve the entire segment, and cut off the part below from the

cerebrum. It is, however, remarkable how much relief is often afforded, even after a prolonged train of disturbances, by removal of the cause, especially when the symptoms point to an affection of the cerebral hemispheres.

As to the nature of the lesion, supposing it to have been determined that it is of syphilitic origin, the most important point is to distinguish between the effect of syphilitic disease in the membranes or nerve substance, and of thrombosis from syphilitic inflammation in the arteries. As Dr. Hughlings Jackson has often insisted, the result of the blocking up of an artery will be independent of the nature of the obstruction. If a collateral supply of blood does not find its way to the part, softening is inevitable; and supposing that treatment could affect the original disease, as when a cerebral artery is included in a gumma, it would probably come too late to obviate the effects. Usually, as has been stated, thrombosis gives rise to sudden attacks without much pain.

Syphilitic epilepsy, so called, yields to treatment. We have here, in Dr. Jackson's language, only a discharging lesion, not a destructive one. In paralysis, on the other hand, there is frequently destruction, but recovery may be expected if we can exclude thrombosis and softening, and if the duration has not been too prolonged. The tumours which give rise to unilateral convulsion appear to be particularly liable to be attended with optic neuritis, and may wear out the patient's strength; but tumours at any part can sometimes be brought to a state of quiescence, and the effects of disseminated lesions are more serious than those produced by a single growth.

As to the seat of the lesion, I will only further add that growths from the dura mater are apparently less amenable to treatment than affections of the other membranes, or of the nervous substance; probably because they are less vascular, and therefore less easily reached and less freely acted upon by the remedies.

The treatment is simple. The one remedy is iodide of potassium; or, this failing, mercury. I usually begin with doses of 6 grains, and always combine with it ammonia—the carbonate or aromatic spirit. Having by one or two days' experience ascertained that there is no special intolerance of the iodide, it may be rapidly pushed to doses of twelve, eighteen, twenty-four, or thirty grains three times a day; occasionally even larger doses are necessary, and I have given a drachm every four hours. That large doses are often absolutely required, and that they succeed when moderate doses fail, I am convinced by abundant experience; and if iodism is induced, which is very rarely the case in tertiary syphilis, it is almost always before large doses are reached. Large doses

are better borne when taken after meals. Of course iodide of potassium is more quickly taken up into the blood from an empty stomach, but it is also quickly out of the blood and in the urine; and when a continuous action on the system is needed, which is what we require in dealing with the effects of tertiary syphilis, the indication is best met by giving so diffusible a remedy as the iodide of potassium after food.

If the iodide of potassium fails after a full and free trial, a resort to mercury is always desirable, and the more recent the syphilis the earlier. When we are passing from the use of one to the other drug, either a certain interval should be allowed to elapse, or the mercury, if given by the mouth, should be in one of its most soluble and active forms—the bichloride or biniodide. More than once I have seen sudden and profuse salivation when this precaution has been neglected, no doubt from the mercury being converted into biniodide within the system. Sometimes I have employed mercurial inunction at the same time with the internal administration of iodide of potassium, and have frequently given biniodide of mercury with iodide of potassium. In many cases I have given a tonic, such as cod-liver oil and iron, or phosphorus, as well as the iodide.

One word as to the *modus operandi* of iodide of potassium. This was the subject of a beautiful explanation by Dr. Odling in his *Gulstonian Lectures*, hypothetical at that time, but demonstrated by experiment since. The active agent is the iodine, as shown by the fact that other salts of potassium have not the same effect, while other combinations of iodine, such as iodide of ammonium or sodium, have. The iodine is permitted to exercise its influence on the seat of disease in virtue of the comparatively slight affinity by which it is held in union by the base, this being so feeble that in the presence of certain forms of living protoplasm in active change the salt is decomposed and the iodine set free to exercise its solvent action on the organic matter; whether this is direct or indirect through the well-known oxidizing effects of free iodine is not so certain.

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